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THE ANNUAL MEETING

SECTION OF PEDIATRICS

TUESDAY MORNING, JUNE 7, 1927

THE Section of Pediatrics convened in the Georgian Room of the Hotel Statler, Boston, Mass., at nine o'clock, Dr. Arthur R. Crandell, of Taunton, presiding.

CHAIRMAN CRANDELL: The first paper this morning will be "The Chemistry Findings in Rickets," which is to be presented by Dr. James L. Gamble, of Boston.

THE CHEMISTRY FINDINGS IN RICKETS

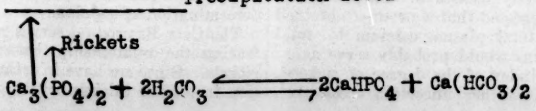
BY JAMES L. GAMBLE, M.D.

RICKETS is a disease which very openly invites study by chemistry methods. The presenting lesion is obviously a chemical one, viz., the failure of deposition of lime salts in the growing ground substance of bone. The problem of rickets was completely defined by Lehnerdt's question, "Warum bleibt das rachitische knockengewebe unverkalkt?" As in all instances of pathological physiology we must first learn if we can what the normal mechanism is. So that here we have an antecedent question,—what are the factors concerned in the precipitation of calcium phosphate in the osteoid tissue under normal circumstances?

This question has not yet been answered by direct data. We have only theory based on in vitro studies of the behaviour in solution of the substances concerned. The most plausible explanation was put forward by Howland and Kramer[1]. They suggested that the precipitation of triple calcium phosphate might be due to a lower concentration of carbonic acid in the interstitial fluid of the osteoid tissue than in the blood plasma. Their theory is described by the following equation:

Here we have carried in the blood plasma triple calcium phosphate and carbonic acid on one side of the equation and on the other side two calcium containing salts, di-calcium phosphate and calcium carbonate. This equation represents a balanced situation. If the balance is disturbed on either side, changes tending to restore it will occur. According to the theory of Howland and Kramer when these plasma substances arrive in the osteoid tissue and diffuse into the interstitial spaces the concentration of carbonic acid falls below its plasma level. The resulting disturbance of balance tends to be restored by increase of the other factor, triple calcium phosphate, at the expense of the concentrations of di-calcium phosphate and calcium carbonate. Triple calcium phosphate, however, is an only slightly soluble substance and is normally carried in the blood plasma at a concentration not far below its saturation level. An increase in its concentration bringing it to this level will result in precipitation to such further extent as it is formed.

If this is the normal mechanism, how may we imagine that precipitation of triple calcium phosphate fails to take place in rickets? Looking at the equation we see two possibilities. Perhaps the lowering of carbonic acid concentration does not occur. We have no evidence which would support such a surmise. A much more probable explanation would be a less than usual amount of the material, di-calcium phosphate and calcium carbonate, from which triple calcium phosphate can be constructed. This we know to be actually true of the blood plasma in rickets and we may therefore expect a less than usual increase in the concentration of triple calcium phosphate in the interstitial fluid of the osteoid tissue. If this increase falls short of the



saturation level precipitation will obviously not occur. This theory is supported by the evidence of very ingenious in vitro experiments in which calcification of slices of epiphyseal cartilage from rachitic rats was obtained according to its terms[2]. We may accept it as probably dependably describing the chief physical factors in the process of calcification.

As regards the lack of material in rachitic plasma for the formation of the lime salts of bone. This finding was anticipated for a long time before it was established. Effort was first centered in the attempt to demonstrate that the blood plasma in rickets carries less than the normal amount of calcium. For a number of years the results of a great many studies were not in agreement owing to unsatisfactory methods for measuring calcium in small samples of blood plasma. Finally, however, as methods became sufficiently accurate, it was established that there is usually little or no lowering of plasma calcium in ordinary rickets. Calcium in blood plasma is normally remarkably stationary at between 10 and 11 mg. per 100 cc. In rickets of the ordinary type it is only rarely found below 9 mg. This was a great disappointment but joy returned when the other equally important factor in the formation of triple calcium phosphate, viz., phosphate ion, was thought of. In 1919 Howland and Kramer[3] and independently Iverson and Lenstrup in Denmark showed that inorganic phosphorus in the blood plasma in ordinary rickets is greatly reduced. Normally the plasma phosphorus of infants is between 5 and 6 mg. per 100 cc. The average value found by Howland and Kramer in a series of 72 cases was 2.5 mg., a 50% reduction. In the same cases the average for plasma calcium was 9.6 mg., a reduction of approximately 5%.

According to the equation given above the triple calcium phosphate deposited in bone is constructed from the di-calcium phosphate and calcium carbonate carried in the plasma. The concentrations of these substances in the molecular form are, according to the rules of physical chemistry determined by the *product* of the concentrations of calcium ions, phosphate ions, and bicarbonate ions in the plasma. Since in rickets the bicarbonate factor is unaltered, the quantity of di-calcium phosphate and calcium bicarbonate in the plasma is determined by the product of the concentrations of calcium and phosphate ions. The value for the product of these two factors should, therefore, inform us of the extent of reduction in the plasma of the material required for calcification. The ionized calcium and phosphate of the plasma can not, however, be directly measured. Howland and Kramer have suggested that a product obtained by multiplying total plasma calcium by total plasma phosphorus would probably serve as an equally reliable index of the degree of rickets[1]. This inference is rather uncertain. However, data which they have published does show a fairly close correlation between the size of the Ca x P. product obtained in this way and the degree of rickets as indicated by x-ray and other clinical evidences. To illustrate the range of this product a few of their data are given in Table 1. As may be seen in the table using the upper and lower levels for normal calcium and phosphorus we obtain a range of product of from 50 to 66 which may be taken as normal. The next three sets of measurements are from infants with severe rickets and the products are found to be below 20. By giving these infants cod liver oil the product was restored to even above the usual value.

Ca	P	Ca x P
	NORMAL	
10	5	50
11	6	66
	RICKETS	
10.5	1.8	19
9.8	1.6	16
9.6	.8	8
	AFTER COD LIVER OIL	
10.2	6.0	61
11.0	6.8	70
10.0	6.0	60
	TETANY	
5.3	5.8	31
5.6	5.1	29
6.7	3.6	24

Table 1. Data from Howland and Kramer (1)

Values for Ca and P. are mg. per 100 cc. of blood serum

Theoretically the Ca x P. product is attractive as a means of measuring the severity of rickets and the effect of therapy. Practically, however, since one of the factors, calcium, remains nearly stationary in ordinary rickets at about the normal value, we are just as well informed by measurement of phosphorus alone. The figures in the table show this point clearly, i. e., the same range of difference in the phosphate measurement as in the product. The reason that the numerals are almost identical is of course that the values for calcium are close to 10. So that if we wish to gauge the degree of ordinary rickets, i. e., rickets uncomplicated by tetany, by what we can find in the blood plasma we need only measure phosphorus. This is a convenience since the measurement of plasma phosphate is a very simple procedure; much more so than the determination of calcium.

The Ca x P. product serves very well in illustrating the relationship between tetany and rickets. Since we have in tetany a large reduction in plasma calcium with usually no or only

slight alteration of phosphorus we must have a much smaller than normal product and we may therefore expect that, when we find tetany, rickets will also be present. Tetany is often described as low calcium rickets and ordinary rickets as the low phosphorus form of the disease. We should not, however, derive the inference that there are two kinds of rickets, that is, in the sense of an independent pathogenesis for each. It is probable that the low calcium form with accompanying tetany is an incident which may occur in the course of ordinary rickets. Gerstenberger[4] has recently presented evidence which suggests that the inversion of the values for calcium and phosphorus seen in tetany may be the result of a brief and inadequate exposure of ordinary rickets to some rickets curative agency.

The lack of phosphate, or occasionally of calcium, in the blood plasma may be regarded as the immediate factor in the failure of calcification of the growing osteoid. What are the initial factors in the pathogenesis of rickets which are responsible for this lack of material in the plasma? As a result of the discovery a few years ago that rickets may be produced in animals by certain diets, this question has been industriously and extensively studied. There has been brisk competition by a great many workers in the attempt to identify the dietary factors responsible for experimental rickets. The information obtained will be only briefly indicated here. A most excellent discussion of the results of this important method of study has been written by Park[5].

Young rats have been the animals chiefly used. Apparently two dietary defects are required for production of rickets. There must be a low, or else an unbalanced, intake of calcium and phosphorus and a lack of an unidentified organic substance usually designated as D. When these dietary conditions have been fulfilled and the young rats have been on the diet for several weeks, it is found that the abnormalities as regards the calcium-phosphorus intake are reflected in the blood plasma, and histological examination of the bones may show active rickets. In Table 2 are given a few data from experiments by Park, Guy and Powers[6] which show a rough correlation between the values found for calcium and phosphorus in the plasma and the amounts of these substances in the diets. It has been found that the low phosphorus-high calcium diet is the most dependable one for the production of experimental rickets. With this diet the high calcium intake tends to completely block the absorption of phosphorus by the formation of difficultly soluble triple calcium phosphate in the intestinal tract. As may be seen in the table, this diet (III) produces in the plasma the large reduction of phosphorus characteristic of human rickets. Attempts to produce the inverse situation, viz., low calcium rickets by high phosphorus-low calcium diet, have not been so definitely successful. According to the data in the table (Diet II), a lowering of calcium is obtained but this is accompanied by a rise in phosphorus of such extent as to produce a normal product of the two factors. The possibility of production of low calcium rickets by dietary defects has been denied by several workers. The table also contains data demonstrating that without changing the diets the administration of cod liver oil, which supplies D abundantly, brings about a remarkably rapid and accurate restoration of the normal values for plasma calcium and phosphorus. The additional large fact which has recently been established is that sunlight produces the same effect as does cod liver oil. If in the above experiments, for example, the rats had been exposed to sunlight for several hours daily instead of being given cod liver oil, an equally rapid return of calcium and phosphorus to normal values would have been obtained. We have thus for contemplation the spectacular fact that the correct adjustment of calcium and phosphorus metabolism produced by ingestion of an unknown substance contained in cod liver oil may also be obtained by permitting sunlight to reach the skin of the body. The inference that the substance D in cod liver oil possesses a photochemical form of activity has been substantiated by the recent important experiments of Hess[7] and of Steenbock[8] demonstrating that various oils ordinarily therapeutically inert may be rendered rickets curative by irradiation. We are thus brought to the threshold of the study of the

CALCIUM AND PHOSPHORUS IN FOOD mg. per 100 gm.			CALCIUM AND PHOSPHORUS IN BLOOD SERUM mg. per 100 cc.			
			No Cod Liver Oil		2% Cod Liver Oil	
Diet	Ca.	P.	Ca.	P.	Ca.	P.
I	0.01	0.29	5.0	6.0	8.8	5.8
II	0.007	0.59	4.2	11.1	8.6	10.8
III	1.42	0.26	10.1	1.3	10.7	6.0

Table 2. Data from experiments by Park, Guy, and Powers[6].

role of photo-chemical reactions in physiological processes. The results of studies in experimental rickets then inform us that lack of a certain unidentified food substance and of sunlight are the chief causative factors in rickets. Of the manner in which these agencies bring about correct concentrations of calcium and especially of phosphate in the body fluids we have as yet no knowledge. The data so far obtained simply demonstrate this control. There is every reason to hope, however, that experimental rickets constitutes a method of study which will ultimately give us a knowledge of the pathogenesis of rickets in full chemical detail.

From the practical point of view the important result of the recent studies of rickets is the rediscovery of the curative action of cod liver oil and sunlight. That rediscovery was necessary in order to persuade us to use these extremely simple and effective agencies is an interesting commentary on the present day method of mind in medicine. That cod liver oil will cure rickets has been known for more than two hundred years. That sunlight is preventive was convincingly evident to students of rickets generations ago. However, with the dawn of the present era of scientific skepticism it became at once evident that the belief that oil from the liver of a codfish is specifically curative of a human ailment must be a bit of old fashioned fetish. To a respectably critical attitude of mind such a proposition was obviously a medical superstition. As a result of this highly valued critical sense, certain upper faculties of mind which might enable us to read indirect evidence correctly have atrophied so we must now have our proof in black and white. It was therefore quite necessary that the curative action of cod liver oil and sunlight be demonstrated again in such actual terms as mg. of calcium and phosphorus. This reëstablishment of our faith in these agencies is a matter of great importance. It should enable us to greatly reduce the incidence of this disease which indirectly underlies so large a part of infant mortality. Naturally as a product of the enthusiasm of renewed faith we have the ardent propagandist who demands that rickets be at once crusaded out of existence. He believes that we should amend the Hippocratic oath to include a promise to give every infant cod liver oil every day. It is to be hoped, however, that we will maintain our intellectual dignity by deciding whether or not the individual infant should be given cod liver oil prophylactically according to such data as the time of year and the infant's opportunities for hygienic care.

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CHAIRMAN CRANDELL: The next paper to be presented is that of Dr. Edwin T. Wyman, of Boston, on "The Prevention and Treatment of Rickets."

THE PREVENTION AND TREATMENT OF RICKETS*

BY EDWIN T. WYMAN, M.D.

ACUTE RICKETS occurring in the first two years of infancy may be defined as a condition in which there is a disturbance in the mineral metabolism of the growing organism. It is of such nature as to disturb the calcium and phosphorus ratio in the tissues so as to prevent normal lime salt deposit in the bones. It occurs during that period of infancy in which active growth is most rapid. The symptoms develop gradually and when well advanced the disease is characterized by restlessness, head sweating, delayed dentition, softening of the skull bone, flabby muscles and characteristic deformities of the bones.

Rickets occurs chiefly in Europe and North America. It is a disease found in cities. It is most prevalent in those nations whose wealth and industrial development have brought about most fully the substitution of artificial conditions of living in place of the simple conditions which nature intended.

Rickets begins to increase in the fall, becomes more marked in the winter and reaches its peak in March; it then gradually decreases and new cases are rarely seen after June.

Now, whereas Glisson in his classical monograph in 1659 established rickets as a clinical entity, and Pommer in 1880 describes, as a result of histological studies, the pathological changes which occur in rickets, but little information was added until it was possible to produce rickets experimentally in animals. The prevention and cure of rickets, it may be said, has been placed on a rational basis from the information resulting directly from these studies.

Mellanby was the first to discover that animals could be fed a diet which would produce rickets. At first dogs were used, but rats have proved to be more practical and are generally used in all metabolism laboratories. Sherman

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and Pappenheimer, McCollum and his co-workers have devised diets for rats which will prevent or cause rickets. The animals are killed and the bones studied for evidences of the disease. Some authorities question the identity of experimental and human rickets, but the bone pathology is much the same. Direct proof of the curative action of cod liver oil was first obtained by McCollum, Simmons, Shipley and Park. These investigators discovered early that cod liver oil caused remarkable depositions of lime salt to form in the cartilage of the rachitic rats. Howland and Park proved by means of X-rays that the administration of cod liver oil to rachitic children was followed by the deposition of lime salts in the cartilage and bones after a period of 15 to 21 days.

During the past few years two remedies have been used extensively in the prevention and treatment of rickets—cod liver oil and ultra violet irradiation. According to Trousseau, cod liver oil has been used from time immemorial as a folk remedy on the coasts of Holland, England and France. It was introduced into France as a specific for rickets by Bretonneau and its use became general through the teachings of Trousseau. Trousseau, himself, was convinced of its curative action in rickets and many physicians have been certain of its effectiveness in rickets purely as a result of clinical observation. However, until definite proof of the curative action of cod liver oil in rickets was brought forth, its beneficial effects were subject to much discussion. It is fairly well admitted now, as the result of recent laboratory studies, that cod liver oil is effective in the prevention and treatment of rickets. In other words, the faith of our forefathers has been reëstablished in cod liver oil.

The experimental study showing the effect of the ultraviolet sun's rays and the artificial rays from the mercury vapor quartz lamp has been very extensive in the last few years and has added greatly to our knowledge of rickets. Huldschinsky first reported in 1920 the beneficial effect of quartz lamp therapy upon rachitic bones. Howland and his associates in 1922 found that the phosphorus in the blood of rachitic children was raised by quartz lamp exposure. Both in experimental and clinical practice these findings have been constantly confirmed. Hess and Weinstock rendered cod liver oil and linseed oil antirachitic by irradiation with ultraviolet rays. This work has been extended to other substances, such as lettuce, growing wheat, cholesterol, phytosterol, flour and milk, the irradiations endowing all with antirachitic properties. It is ineffectual on solutions of chlorophyl, red blood cells, cream and phosphatid of egg yolk and glycerine. Hess explains the activation of cholesterol and its value in the treatment and prevention of rickets as follows: the epidermal portions of the skin contains a large amount of cholesterol in its deeper layers, which is probably rendered active by the sun or similar artificial rays. This hypothesis presupposes the formation of active cholesterol within the skin and its transport through the body by the circulation.

These two therapeutic measures—cod liver oil and ultraviolet irradiation—are recognized now as being almost specific in action when used to prevent and cure active or infantile rickets.

Another specific measure has lately been claiming attention,—the direct irradiation with the sun's rays through ultraviolet transmitting glasses. The principle upon which such glass depends is as follows:—Light waves from the sun extend into the ultraviolet region to 290 millimicrons. All shorter waves are absorbed in passing through the atmosphere. The rays from this limit, 290 millimicrons to 310 millimicrons, have been shown to be the beneficial rays in the cure of rickets. These ultraviolet rays in the sun's spectrum vary in intensity during the different seasons of the year, being highest during the summer months and lowest during the winter months. They are diminished in low altitudes and are filtered out by dust and moisture in the air.

A natural objection to the use of this means of therapy was raised on account of the geographical location of Boston. It was questionable whether or not sufficient antirachitic radiation could be obtained from the solar spectrum through quartz and other ultraviolet transmitting glasses to be effective in curing rickets during the winter months. The average total hours of monthly sunshine in Boston as recorded by the United States Department of Agriculture Weather Bureau for the past ten years are: January 142.4 hours, February 169.5 hours, March 211.8 hours, April 220.6 hours, May 274.6 hours, June 286.3 hours, July 305 hours, August 269.6 hours, September 228.2 hours, October 194.3 hours, November 141.8 hours and December 123.2 hours. (See Figure 1.) The total

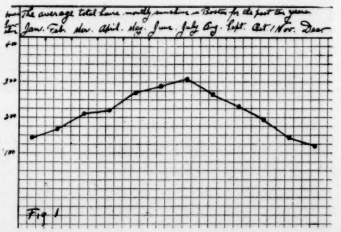


Fig. 1.

hours of monthly sunshine are shown more in detail in Table No. 1. It will be seen that the

U. S. DEPARTMENT OF AGRICULTURE, WEATHER BUREAU

Station, Boston, Mass.

Date: Total Hours of Sunshine

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total
1917	119	171	224	204	216	287	296	307	207	166	193	112	2649
18	168	170	248	250	311	304	271	264	207	168	148	110	2808
19	121	207	173	193	276	230	301	260	195	134	125	126	2334
20	147	143	237	165	252	230	243	246	225	220	88	152	2465
21	163	169	210	174	266	332	274	240	249	224	94	132	2659
22	172	126	178	248	232	251	294	224	252	214	139	90	2514
23	116	160	193	241	291	261	250	236	252	200	145	118	2503
24	162	199	224	224	225	299	247	275	222	251	171	122	2721
25	141	186	210	241	274	211	301	234	209	145	140	124	2996
26	104	126	211	234	283	270	248	300	218	194	160	130	2401
27	125	118	224	242	217								

AVERAGE MONTHLY SUNSHINE FOR TEN YEARS

1917 to 1926, Inclusive.

Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
Hrs. 142.4	149.5	211.0	230.6	274.5	266.5	305.0	269.6	220.2	194.3	141.6	123.8

TABLE I

average daily sunshine is 3.97 hours during the month of December and 9.83 hours during the month of July.

Infants do not develop rickets in the summer on account of the protective effect of the sun's rays but in this latitude under usual winter conditions of housing, climate and clothing, the direct radiations that reach the growing baby do not suffice to prevent its development. It is impracticable to expose infants to the direct rays of the sun except in the warm summer months on account of the cold and consequent loss of body heat. We therefore determined to find out if it was possible to obtain sufficient ultraviolet rays through quartz and other ultraviolet transmitting glasses during the winter months to be effective in curing rickets.

The windows used in this experiment were clear quartz 1/8 of an inch thick, manufactured by the General Electric Company, Corning glass G980 6 to 9 mm. in thickness, manufactured by the Corning Glass Works, Corning, N. Y., and Lamplough's Vitaglass (cathedral type) 3 mm. in thickness, an English product. The ordinary glass used in windows and sun porches transmits a portion of the ultraviolet nearest the visible region of the spectrum but entirely filters out the vital rays between 290 and 310 millimicrons. Quartz glass is very transparent to the shorter rays in the sun's spectrum. Tests on the transparency of the Corning glass and the Vitaglass used in these windows to the rays from the mercury vapor arc were made by Dr. Donald C. Stockbarger at the Massachusetts Institute of Technology, and are shown in the spectrogram. (See Figure 2.) It will be seen that the Corning glass transmits rays down to 265 millimicrons and the Vitaglass transmits down to 290 millimicrons, the extreme limit of the sun's spectrum. Of the two samples of ordinary window glass, one transmitted rays down to 312 millimicrons and the other than 333 millimicrons. A number of carefully controlled observations were made on infants suffering with acute rickets. The babies were placed directly in front of windows composed of these various types of glass. (See Figures 3 and 4.) They remained undressed all day so as to receive the benefit of all the available sunshine possible and careful notes were kept of actual length of time they were in the sunshine each day. Curtains were placed in front of the cubicles and heaters used when necessary to maintain a room temperature of 80°F. The windows were not ideally situated as will be seen by the hours of direct sunlight received by the patients. The babies were kept as nearly as possible on the same diet they received on admission to the hospital with the exception of increasing the food elements as they gained weight, in order to meet their caloric requirements. We have treated nine cases in this manner. Four with the sun's rays through quartz, three through Corning glass and two through Vitaglass. Three babies with severe

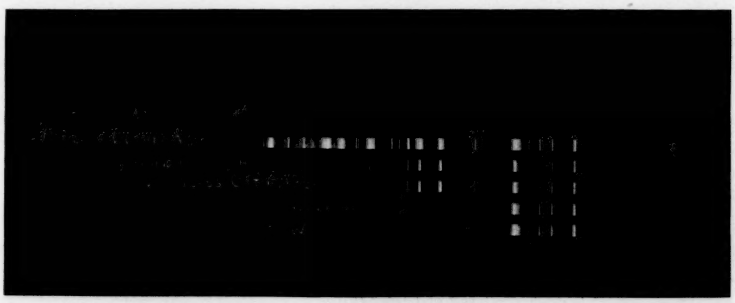


FIGURE 2. Ultraviolet transmission spectra of Corning glass, Vitaglass and ordinary window glass.

FIGURE 3. Baby receiving treatment with sunlight through quartz window.

FIGURE 4. Babies receiving treatment with sunlight through Corning glass and Vitaglass windows.

rickets treated last year in front of the quartz window made rapid recovery. Though none of these infants had had a history of having cod liver oil or other antirachitic substances before admission, there was the possibility of their having had some antirachitic substance in their diet before they came under observation in the hospital which might have had more or less influence on their rapid recovery. In the experiments carried on the past winter, the infants have been kept under observation for from ten to twenty days without antirachitic therapy before the treatment was started, to overcome this possibility. All the patients treated with sunlight through the quartz and Corning glass showed definite evidence of recovery as determined by the clinical evidence, roentgenogram and blood examination. The results in the two cases treated by sunlight through Vitaglass were disappointing. Both these patients were very black negroes and it is possible that babies with lighter complexions would have been benefited. The results obtained with Vitaglass, however, did not compare with similar cases treated with the sun's rays through quartz and Corning glass.

Table No. 2

Case No. 1 L.H. Colored female infant aged 10 1/2 months.

RECORD OF SUNLIGHT RECEIVED THROUGH QUARTZ WINDOW 12-17-25

Week	Days of Sunshine	Total Exposure	Approximate time of day
1st 12-17-25 to 12-23-25	3 days	7 hours 40 mins.	9 a.m. to 1 p.m.
2nd 12-24-25 to 12-30-25	5	21	8:30 a.m. to 1:15 p.m.
3rd 12-31-25 to 1- 6-26	5	22 45	8:30 a.m. to 1:15 p.m.
4th 1- 7-26 to 1-13-26	5	22 45	8:30 a.m. to 1:30 p.m.
5th 1-14-26 to 1-20-26	5	20 00	8:30 a.m. to 1:30 p.m.
6th 1-21-26 to 1-27-26	4	14 30	10 a.m. to 1:45 p.m.
7th 1-28-26 to 2- 3-26	3	10 00	9:30 a.m. to 2:30 p.m.
8th 2- 4-26 to 2-10-26	5	18 40	9:45 a.m. to 2:00 p.m.

Total hours of sunshine through quartz window 136 hours 40 minutes, a daily average of 2 hours and 20 minutes.

Case No. 1 L.H.

BLOOD EXAMINATION

	Hemoglobin	Red Blood Count
12-17-25	70%	15,000
1-11-26	70%	8,500
1-21-26	70%	11,000

	Calcium	Phosphorus	Product of Ca. and P
12-16-25	10.8	3.1	33.4
12-31-25	10.4	3.2	33.2
1-7-26	9.0	3.8	34.2
1-14-26	8.8	Lost	
1-21-26	8.9	4.17	36.8
1-30-26	10.4	3.8	39.5
2- 5-26	10.7	4.16	44.5
2-14-26	11.2	4.9	54.8

Case 1. L. H., a colored, female infant, 10½ months old. She was admitted to the Infants' Hospital December 17, 1925, and discharged February 14, 1926. The family history was negative.

She was a premature baby, born at the seventh month. Instruments and ether were employed at delivery. Her birth weight was 7½ pounds. She was breast fed until three months of age. After that she was given a whole milk mixture with Dextri-Maltose added. The formula was gradually increased until. at the time of admission, she was taking

Whole milk, 32 ounces
Boiled water, 14 ounces
No. 1 Dextri-Maltose, 1 level tablespoonful

In addition to the formula she was also taking cereal, potato, carrots, orange juice, baked apple and bread.

The chief complaints were failure to use her legs and head sweating.

Physical Examination: She was a poorly developed and fairly well nourished negro baby, lying quietly with no apparent pain. She sat up with some help and held head well, but made little effort to use lower extremities. She had all the physical signs of severe rickets — prominent frontal and parietal bosses, anterior fontanelle larger than normal, epiphyses at the wrists and ankles markedly enlarged and craniotabes. Harrison's groove was marked. There was a well defined rosary and lower rib margin was flaring. The abdomen was prominent. The spleen and liver were both palpable, the former 4 cm. below the costal margin and the latter 2 cm. below the costal margin.

The diagnosis of Acute Rickets was made.

The blood examination showed a calcium concentration of 10.8 milligrams of calcium per 100 c.c. and a phosphorus of 3.1 milligrams per 100 c.c. of serum.

Roentgenograms of the wrist bones showed the marked changes of active rickets.

The Wassermann was negative and the tuberculin test was negative.

She was placed in front of the quartz window, stripped, with the exception of a diaper, and she was given all the sunshine obtainable.

Her diet was continued the same as before admission and she took from 60 to 65 calories per pound during her stay in the hospital.

She was discharged on February 14. Her weight had increased from 12 pounds on admission to 13 pounds and 4 ounces on discharge. She received a total of 136 hours and 40 minutes of sunshine, a daily average of 2 hours and 20 minutes. (See Table No. 2.)

During her stay in the hospital the product of the serum calcium and the phosphorus increased from 33.4 to 54.88. (See Table No. 2.) The hemoglobin was 75% and during the stay in the hospital and the red count increased from 3,360,000 to 4,410,000.

Roentgen-ray examination showed progressive increase in the density of the epiphyses and the shafts of the bones. The last roentgenogram taken showed the process well healed. (See Figure No. 5.)

The craniotabes disappeared along with other symptomatic evidences of acute rickets.

Case 2. J. C., a white, male infant of 4 months. Admitted to the Infants' Hospital, February 11, 1926, and discharged March 28, 1926. The family history was negative.

He was a full term baby born after an instrumental delivery. His birth weight was 8-5. He was breast fed to 22 days, then put on a formula consisting of

Whole milk, 13½ ounces
Water, 7½ ounces
Karo Corn Syrup, 2 ounces

He gained well on this up to 3½ months, two weeks before admission. His weight then remained stationary. Two days before admission the formula was changed to

28 ounces whole milk
14 ounces water
7 tablespoonfuls of Dextri-Maltose No. 2

Three weeks before admission he had much difficulty in breathing and a crowing sound on inspiration. These symptoms had continued up to the time of admission. The attacks of crowing occurred four or five times a day and were accompanied by cyanosis. They became increasingly frequent in number and duration until the most severe one, which occurred the morning before admission and lasted 20 minutes. It was accompanied by extreme cyanosis and respiratory difficulty. During this atttack the arms and legs became stiff, the eyes were wide open and he was unconscious.

Physical Examination: He was a well developed, undernourished baby. His color was pale. He was quiet and conscious. He had a marked inspiratory crow when he cried and breathed somewhat rapidly. The head showed marked craniotabes and the anterior fontanelle was widely open. The posterior fontanelle was patent. The chest showed a marked rachitic rosary and some evidence of Harrison's groove. The abdomen was distended. The liver was 3 cm. below the costal margin and the spleen was just palpable. The reflexes were all active. Chvostek and tibial were not obtained. There was a suggestion of the presence of Trousseau's sign. The electrical reactions were typical of those found in spasmophilia. The Wassermann test and the tuberculin test were negative.

The diagnosis of Spasmophilia and Severe Rickets was made.

He was put in front of a quartz window, stripped, with the exception of a diaper, and given all the sunshine obtainable. He was kept there from the time of entry and was given no other treatment.

During the stay in the hospital the product of the serum calcium and phosphorus increased from 51.84 to 75. (See Table No. 3.) The hemoglobin and red count remained practically the same. The hemoglobin was 69% and the red count around 3,500,000.

Roentgen-ray examination on admission showed a marked degree of active rickets. Subsequent roentgenograms showed gradually increasing density of shafts of the bones and increased density of the epiphyses. The last roentgenogram showed the rachitic process well healed. (Figure No. 6.)

He received a total of 110 hours and 40 minutes of sunshine (see Table No. 3), a daily average of 2 hours and 24 minutes during his stay in front of the quartz window. On the fifth day of exposure he received 5 hours of sunlight and had a definite sunburn.

The weight increased from 12 pounds and 10 ounces to 13 pounds and 14 ounces on discharge.

On entering, a formula of whole milk with Karo corn syrup added, was given to the baby. He took from 50 to 55 calories per pound daily.

The laryngismus stridulus and other signs of spasmophilia disappeared within 10 days after the treatment was started. He had no convulsions after the treatments were started.

FIGURE 5. Case No. 1. L. H. Female aged 10½ months treated with the sun's rays through quartz window panes. Roentgenograms show progressive increase in the density of the epiphyses and shafts of the bones.

CASE 3. R. J. was a 23 months-o'd white child brought to the hospital January 26, 1927, and discharged April 23, 1927. He was a full term baby born after a fairly difficult labor and weighed 8¾ pounds. He was breast fed for two months, and was then given a whole milk, water and sugar mixture of unknown amounts. His diet on admission consisted of milk, cereal and vegetable.

Physical Examination: He was a small undernourished child with marked beading of the ribs.

Table No. 3

Case No. 2 J. C. White male infant aged 4 months.

RICKAD OF SUNSHINE ADMITTED THROUGH QUARTZ WINDOW 2-11-26

Week	Days of Sunshine	Total Exposure	Approximate time of day
1st 2-11-26 to 2-17-26	5 days	15 hours 25 mins.	10:30 a.m. to 1:30 p.m.
2nd 2-18-26 to 2-24-26	5	16 5	10:30 a.m. to 2 p.m.
3rd 2-25-26 to 3- 3-26	5	11 50	11 a.m. to 1 p.m.
4th 3- 4-26 to 3-10-26	6	21 10	11 a.m. to 2:30 p.m.
5th 3-11-26 to 3-17-26	5	20 15	11:15 a.m. to 3:15 p.m.
6th 3-18-26 to 3-24-26	4	19 20	11:40 a.m. to 4 p.m.
7th 3-25-26 to 3-27-26	2	6 35	1:30 p.m. to 4:30 p.m.

Discharged the middle of seventh week from hospital.

Total hours of sunshine received through quartz window 110 hours and 40 minutes, a daily average of 2 hours and 24 minutes.

Case No. 2 J. C.

BLOOD EXAMINATION

	Hemoglobin	Red Blood Count
2-23-26	69%	3,840,000
2- 2-26	69%	3,784,000
3- 6-26	68%	3,960,000
3-16-26	69%	3,960,000
3-21-26	69%	3,080,000

	Calcium	Phosphorus	Product of Ca. and P.
2-11-26	6.4	8.2	51.8
2-19-26	7.4	5.80	44.
3- 2-26	7.4	8.24	61.7
3-11-26	10.3	6.4	65.9
3-21-26	12	6.25	75.

He had enlarged frontal and parietal eminences. The epiphyses of the wrists and ankles were enlarged. The tuberculin and Wassermann tests were negative. The roentgen-ray examination of the wrists and the calcium and phosphorus determination of the blood confirmed the diagnosis of rickets.

After admission on January 26 he was kept under observation for 21 days without antirachitic therapy and at the end of that time there was no evidence of healing and the product of the calcium and phosphorus was 38.4 mg. per 100 c.c. of serum as compared with 36 mg. per 100 c.c. on admission. (See Table No. 4.)

He was placed in front of a Corning glass window on February 15 and received a total of 110 hours and 45 minutes sunshine, a daily average of 1 hour and 40 minutes. (See Table No. 4.)

His convalescence was steady and fairly rapid, as shown by the roentgenograms. (See Figure No. 7.)

He weighed 19¾ pounds at time of discharge on April 23, a gain of 1 pound and 4 ounces over his admission weight.

Case 4. D. I. was an 18-months-old negro infant. She was admitted to the hospital on February 16, 1927. She was born at full term after a normal delivery. She was normal at birth and weighed 7 pounds. She was breast fed 5 weeks, after which she had a whole milk modification of unknown proportions as the baby had been "boarded out" a greater

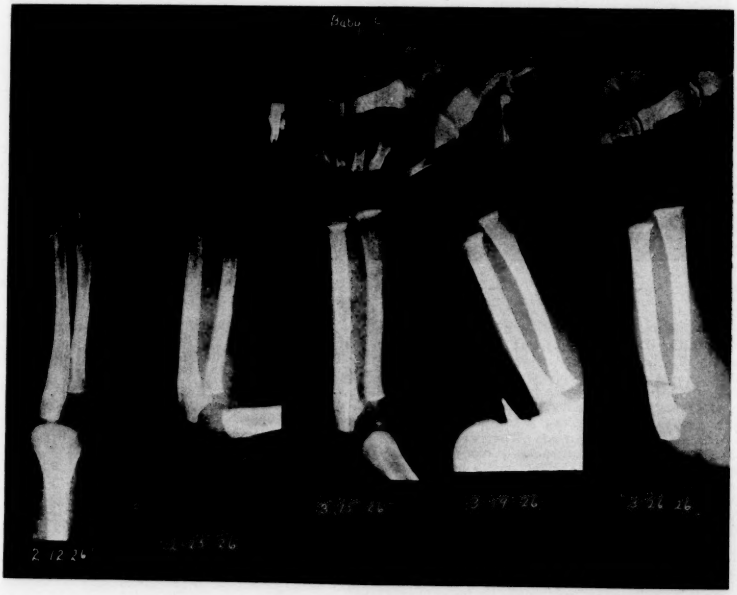


FIGURE 6. Case No. 2. J. C. Male aged 4 months treated with the sun's rays through quartz window panes. Roentgenograms show progressive increased calcification of the epiphyses and shafts with the appearance of the carpal centers of ossification

Table No. 4

Case No. 3 R. J. White male aged 23 months.

RECORD OF SUNSHINE RECEIVED THROUGH CORNING GLASS WINDOW 2-12-27

Week	Days of Sunshine	Total Exposure	Approximate time of day
1st 2-12-27	1	3 hours	12;30 p.m. to 3;30 p.m.
2nd 2-13-27 to 2-20-27	2	7 15 mins.	10;40 a.m. to 3;30 p.m.
3rd 2-21-27 to 2- 1-27	6	23 10	11;30 a.m. to 4 p.m.
4th 3- 2-27 to 3- 8-27	5	13 45	11;30 a.m. to 4;15 p.m.
5th 3- 9-27 to 3-15-27	5	12	11;45 a.m. to 4;50 p.m.
6th 3-16-27 to 3-22-27	4	10 15	12;30 a.m. to 4 p.m.
7th 3-23-27 to 3-29-27	Not exposed		
8th 3-30-27 to 4- 5-27	2	3 45	1;15 p.m. to 4 p.m.
9th 4- 6-27 to 4-12-27	6	12 20	12;45 p.m. to 4 p.m.
10th 4-13-27 to 4-19-27	5	8 15	1;15 p.m. to 3;35 p.m.
11th 4-20-27 to 4-22-27	1	1	1;15 p.m. to 2;15 p.m.

Total hours of sunshine through Corning glass window 110 hours 45 minutes, a daily average of 1 hour and 40 minutes.

Case No. 3 R. J.

BLOOD EXAMINATION

Date	3-23-27	3-18-27	3-30-27	4-14-27
Hgb.	69%	71%	71%	72%
R.B.C.	3,712,000	3,704,000	3,696,000	3,720,000
W.B.C.	9,600	9,000	8,000	8,400
Polys. Neut.	36%	37%	39%	37%
Eos.	2%	3%	1%	2%
Baso.	1%		1%	
Lymph. Small	53%	56%	54%	55%
Large	2%	1%	2%	1%
At.	1%			
Monos.	5%	3%	3%	5%

	CALCIUM	PHOSPHORUS	PRODUCT OF CA. AND P.
1-27-27	10.2	Lost	
1-31-27	9	4.	36.
2-14-27	8.3	4.6	38.4
3- 2-27	9.8	2.7	26.4
3-16-27	10.2	3.41	34.7
4-12-27	10.6	4.8	51.8
4-22-27	9.81	4.	38.

part of the time. At 10 months she was given spinach, squash and wheatena in addition to the milk. For the past two months she had not slept well, had a poor appetite and her head perspired a great deal, especially at night. Her mother noticed the enlargement of the wrists and ankles and for that reason brought her to the hospital.

Physical Examination: She was a rather poorly developed and nourished infant, showing all the clinical signs of rickets, enlarged bones, flexible sutures, marked rosary, Harrison's groove, palpable spleen, prominent abdomen and epiphyseal enlargement.

The laboratory data were negative with the exception of the blood. The blood examination and roentgen-ray examination indicated active rickets.

She was kept under observation without antirachitic therapy for 12 days. On February 12, 1927, she was placed in front of the Vitaglass window. From February 28 to April 11 she received 84 hours and 35 minutes of sunshine, an average of 1 hour and 58 minutes a day. During this time the product f the calcium and phosphorus progressively decreased and the roentgenograms showed no evidence of healing. (See Table No. 5 and Figure No. 8.)

She was placed in front of the Corning glass win-

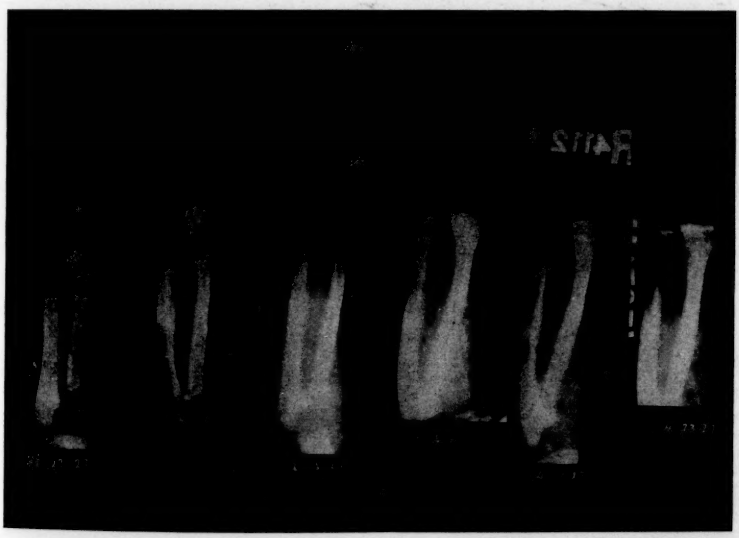


FIGURE 7. Case No. 3. R. J. Male aged 23 months treated with the sun's rays through Corning glass. Roentgenograms show no evidence of healing during the period of observation without antirachitic treatment January 27 to February 21. Then progressive increased calcification is seen.

FIGURE 8. Case No. 4. D. I. Female aged 18 months treated with the sun's rays through Vitaglass. Roentgenograms show very little or no evidence of healing from February 17 to April 11 and slightly increased calcification from April 11 to April 25.

dow in the same room on April 12 and during the next two weeks she received 25 hours and 40 minutes of sunshine, an average of 1 hour and 57 minutes a day. (See Table No. 5.)

The roentgenogram taken on April 25 showed considerably more calcium deposition than the previous one, and there had been a slight increase in the product of the serum calcium and phosphorus. It was thought best to hasten her recovery and she was started on cod liver oil and lamp treatment.

Table No. 5

Case No. 4 D. I. Colored female aged 18 months.

RECORD OF SUNSHINE RECEIVED THROUGH VITAGLASS WINDOW 2-26-27

Week	Days of Sunshine	Total Exposure	Approximate time of day
1st 2-26-27 to 3- 1-27	2	5 hours	11:05 a.m. to 4:30 p.m.
2nd 3- 2-27 to 3- 8-27	6	12 55 mins.	11:10 a.m. to 4:10 p.m.
3rd 3- 9-27 to 3-15-27	4	17 50	11 a.m. to 4:30 p.m.
4th 3-16-27 to 3-22-27	3	11 45	11:30 a.m. to 4:15 p.m.
5th 3-23-27 to 3-27-27	2	6	1 p.m. to 4. p.m.
6th 3-30-27 to 4- 5-27	3	14 20	10:40 p.m. to 4:30 p.m.
7th 4- 6-27 to 4-11-27	6	16 45	12:30 p.m. to 4:30 p.m.

Total hours of sunshine through Vitaglass window 84 hours and 35 minutes, a daily average of 1 hour and 58 minutes.

RECORD OF SUNSHINE RECEIVED THROUGH GLASS WINDOW 4-12-27

Week	Days of Sunshine	Total Exposure	Approximate time of day
7th 4-12-27	1	1 hour 45 mins.	2:45 p.m. to 4:30 p.m.
8th 4-13-27 to 4-19-27	6	19 55	11:20 a.m. to 4 p.m.
9th 4-20-27 to 4-24-27	2	4	2:30 p.m. to 4:30 p.m.

Total hours of sunshine through Corning glass window 25 hours 40 minutes, a daily average of 1 hour and 57 minutes.

Case No. 4 D. I.

BLOOD EXAMINATION

Date	2-21-27	3-8-27	3-10-27	3-30-27	4-11-27	4-21-27
Hgb.	49%	50%	50%	50%	51%	51%
R.B.C.	2,800,000	2,832,000	2,824,000	2,840,000	2,832,000	2,824,000
W.B.C.	6,400	7,800	6,900	8,200	6,800	7,200
Polys. Neut.	38%	36%	29%	40%	37%	38%
Eos.	2%	3%	1%		2%	1%
Baso.		1%				1%
Lymph. Sm.	58%	58%	54%	58%	56%	58%
Large	1%		2%	1%		1%
At.	1%	1%		1%		1%
Mono.	2%	4%	4%	3%	5%	3%

	CALCIUM	PHOSPHORUS	PRODUCT OF CA. AND P.
2-17-27	10.	3.8	38.
3- 1-27	8.8	4.2	36.9
3-14-27	9.6	2.68	25.7
3-28-27	9.1	2.1	19.1
4-11-27	10.	2.56	25.8
4-25-27	10.2	2.9	29.6

During her stay in the hospital she was given the regular hospital diet, with the exception of egg, for an 18-months-old infant.

She was discharged April 28 weighing 17 pounds and 12 ounces.

Charts of the other five patients treated are shown in Tables Nos. 6, 7, 8, 9 and 10.

Table No. 6

Case No. 5 D. U. White female aged 5 months.

RECORD OF SUNSHINE RECEIVED THROUGH ORDINARY WINDOW 4-8-26

Week	Days of Sunshine	Total Exposure	Approximate time of day
1st 4- 8-26 to 4-13-26	4 days	10 hours 35 mins.	1;15 p.m. to 3;30 p.m.
2nd 4-14-26 to 4-20-26	7	15 30	1;20 p.m. to 3;30 p.m.
3rd 4-21-26 to 4-27-26	7	13 40	1;25 p.m. to 3;30 p.m.
4th 4-28-26 to 5- 4-26	4	6 55	2 p.m. to 3;45 p.m.

Total hours of sunshine through ordinary window 46 hours, 40 minutes.

RECORD OF SUNSHINE RECEIVED THROUGH QUARTZ WINDOW 5-5-26

Week	Days of Sunshine	Total Exposure	Approximate time of day
1st 5- 5-26 to 5-11-26	5 days	7 hours	4 p.m. to 5;15 p.m.
2nd 5-12-26 to 5-18-26	3	2 45 mins.	4;30 p.m. to 5;15 p.m.
3rd 5-19-26 to 5-25-26	5	4 30	4;45 p.m. to 5;45 p.m.
4th	No Sun		

Total hours of sunshine through quartz window 14 hours 35 mins.

Table No. 7

Case No. 6 R. W. Colored male aged 16 months.

RECORD OF SUNSHINE RECEIVED THROUGH CORNING GLASS WINDOW 2-23-27

Week	Days of Sunshine	Total Exposure	Approximate time of day
1st 2-23-27 to 3- 1-27	4	9 hours 45 mins.	11;05 a.m. to 4;30 p.m.
2nd 3- 2-27 to 3-10-27	6	16 20	11;30 a.m. to 4 p.m.
3rd 3-11-27 to 3-17-27	6	20 30	11;30 a.m. to 4;30 p.m.
4th 3-18-27 to 3-24-27	3	10	11;30 a.m. to 4 p.m.
5th 3-25-27 to 3-31-27	7	19	1 p.m. to 4 p.m.
6th 4- 1-27 to 4- 7-27	5	20	10;40 a.m. to 4;30 p.m.
7th 4- 8-27 to 4-14-27	6	14 10	2;00 p.m. to 4;30 p.m.
8th 4-15-27 to 4-21-27	3	11 15	11;30 a.m. to 4;15 p.m.
9th 4-22-27 to 4-28-27	1	2	2 p.m. to 4 p.m.

Total hours of sunshine through Corning glass window 123 hours.

Without the advice and help of Dr. Kenneth Blackfan and the assistance of Dr. Gamble's laboratory at the Children's Hospital, these studies would not have been possible.

I wish also to acknowledge my indebtedness to Dr. Joshua E. Bacon and Dr. Robert N. Ganz for their valuable assistance in carrying out these studies, to Dr. A. S. Small for the blood counts, and to Miss H. I. Henry, R.N., and Miss Katherine McKenzie, R.N., for their interest and efficient care of these infants while they were in the hospital. I wish also to thank the Ladies' Aid for their financial help.

From a practical point of view the benefits of our newer knowledge regarding the pathogenesis and therapy of rickets may be summarized as follows:

In the individual infant the need for antirachitic measures should be determined only after careful consideration of the adequacy of diet and the amount of sunshine received.

Case No. 5 D. U.

BLOOD EXAMINATION

	Hemoglobin	Red Blood Count
4-13-26	68%	4,488,000
4-20-26	69%	4,360,000
4-27-26	69%	4,288,000
5- 4-26	69%	4,240,000
Quartz Window 5-5-26 to 5-11-26	69%	4,230,000

	Calcium	Phosphorus	Product of Ca. and P.
4- 8-26	12.8	4.26	52.4
4-20-26	10.5	4.15	43.5
5- 5-26	9.8	4.65	44.6
5-25-26	10.5	6.96	72-9

ROENTGEN-RAY REPORT

The roentgen-ray studies showed no evidence of healing while patient was in front of the ordinary window glass. There was progressive healing after patient was placed in front of the Corning glass window.

Case No. 6 R. W.

BLOOD EXAMINATION

Date	2-23-27	3-6-27	3-16-27	3-30-27	4-14-27	4-24-27
Hgb.	59%	60%	60%	60%	61%	62%
R.B.C.	3,760,000	3,800,000	3,792,000	3,808,000	3,824,000	3,848,000
W.B.C.	9,400	8,800	6,800	7,400	8,200	7,400
Polys. Neut.	38%	37%	39%	40%	37%	37%
Eos.	2%	1%	2%	1%	2%	1%
Baso.		1%				
Lymph. Sm.	58%	56%	54%	58%	58%	57%
Large			1%			
At.					1%	
Monos.	2%	5%	4%	4%	4%	5%

	CALCIUM	PHOSPHORUS	PRODUCT OF CA. AND P.
2-23-27	9.7	3.8	36.8
3- 1-27	9.1	4.65	42.3
3-14-27	9.3	4.05	39.6
3-28-27	10.4	4.55	47.3
4-11-27	9.9	4.44	43.9
4-26-27	9.5	4.42	41.9

ROENTGEN-RAY REPORT

The roentgen-ray studies showed progressive healing during the patient's stay in front of the Corning glass window.

The prevention of rickets in the individual child is a much simpler matter than the public health problems of rickets prevention. Marked symptoms of rickets will seldom be seen in the child who is examined at sufficiently close intervals and given the opportunity for a complete dietary, for the prevention of many infections, and for plenty of fresh air and hours of sunshine each day.

Cod liver oil prevents severe rickets with certainty in the majority of cases. The mild form of rickets which can be made out only by X-ray

in rapidly growing breast fed infants whether or not they are taking cod liver oil, may be a physiological process and is of little consequence.

Table No. 8

Case No. 7 H. R. Colored male aged 2 months.

RECORD OF SUNSHINE RECEIVED THROUGH QUARTZ WINDOW 1-18-27

Week	Days of Sunshine	Total Exposure	Approximate time of day
1st 1-18-27	1	3 hours 40 mins.	11 a.m. to 2:40 p.m.
2nd 1-20-27 to 1-26-27	3	12 15	10:30 a.m. to 2:50 p.m.
3rd 1-27-27 to 2- 2-27	5	12 35	11 a.m. to 3 p.m.
4th 2- 3-27 to 2- 9-27	6	19 40	11 a.m. to 3 p.m.
5th 2-10-27 to 2-16-27	6	13 5	11;10 a.m. to 3:30 p.m.
6th 2-17-27 to 2-23-27	2	8 40	10;40 a.m. to 4 p.m.
7th 2-24-27 to 3- 2-27	6	21 35	11;30 a.m. to 3;30 p.m.
8th 3- 3-27 to 3-9- 27	5	11 55	1 p.m. to 4 p.m.
9th 3-10-27 to 3-16-27	4	9 10	1 p.m. to 4 p.m.
10th 3-17-27 to 3-23-27	4	8 30	1;30 p.m. to 4 p.m.
11th 3-24-27 to 3-30-27	4	9	1;30 p.m. to 4 p.m.
12th 3-31-27 to 4-6-27	3	5 30	1;30 p.m. to 4;15 p.m.
13th 4- 7-27 to 4-13-27	5	12 20	1;30 p.m. to 4 p.m.
14th 4-14-27 to 4-20-27	4	7 40	1;15 p.m. to 4;15 p.m.
15th 4-21-27 to 4-27-27	2	3	1;15 p.m. to 4;15 p.m.

Total hours of sunshine through Quartz window 159 hours 35 minutes.

Case No. 7 H. R.

BLOOD EXAMINATION

Date		1-5-27	1-13-27	1-25-27	2-15-27	3-8-27	3-18-27
Hgb.		80%	80%	80%	80%	80%	81%
R.B.C.		4,480,000	4,528,000	4,888,000	4,400,000	4,488,000	4,352,000
W.B.C.		7,800	8,400	8,800	8,200	7,800	8,200
Polys.	Neut.	40%	37%	39%	36%	39%	36%
	Eos.		2%	1%	2%		3%
	Baso.	1%	1%				
Lymph.	Sm.	54%	58%	54%	56%	55%	53%
	Large	2%		1%		1%	2%
	At.	1%		2%			
	Mones.	2%	2%	3%	4%	5%	4%

Date		3-30-27	4-14-27	4-24-27
Hgb.		80%	81%	82%
R.B.C.		4,380,000	4,368,000	4,352,000
W.B.C.		7,600	8,000	8,800
Polys.	Neut.	39%	36%	37%
	Eos.		3%	3%
	Baso.		1%	
	Sm.	58%	58%	58%
	Large	1%	1%	1%
	At.	2%	1%	1%
	Mones.	2%	3%	3%

	CALCIUM	PHOSPHORUS	PRODUCT OF Ca. AND P.
1- 8-27	9.0	3.9	35.1
1-17-27	9.2	3.53	32.4
1-12-27	9.	3.4	30.6
1-31-27	9.1	4.2	38.2
2-14-27	9.2	4.75	43.7
3- 1-27	8.4	4.95	41.5
3-14-27	9.2	3.36	32.9
3-28-27	10.3	4.4	45.3
4-11-27	10	4.01	40.1
4-25-27	10.7	5.6	59.9

ROENTGEN-RAY REPORT

The roentgen-ray studies showed progressive healing while patient was having sunlight through the quarts window.

Table No. 9

Case No. 8 A.F. White male aged 9 months.

RECORD OF SUNSHINE RECEIVED THROUGH CORNING GLASS WINDOW 2-10-27

Week	Days of Sunshine	Total Exposure	Approximate time of day
1st 2-10-27 to 2-16-27	2	5 hours 10 mins.	12;10 p.m. to 3;40 p.m.
2nd 2-24-27 to 3- 2-27	2	6 15	12;10 p.m. to 3;40 p.m.
3rd 3- 3-27 to 3- 9-27	4	10 35	12;30 p.m. to 4;10 p.m.
4th 3-10-27 to 3-16-27	5	15 40	12 n. to 4;15 p.m.
5th 3-17-27 to 3-23-27	4	10	1 p.m. to 4 p.m.
6th 3-24-27 to 3-30-27	6	13 30	1;30 p.m. to 4 p.m.
7th 3-31-27 to 4- 6-27	4	6 15	1;30 p.m. to 4 p.m.
8th 4- 7-27 to 4-13-27	7	13 30	12;45 p.m. to 4 p.m.
9th 4-14-27 to 4-20-27	3	3 45	3 p.m. to 4;30 p.m.
10th 4-21-27 to 4-27-27	1	5	2;30 p.m. to 4 p.m.

Total hours of sunshine through Corning glass window 91 hours 40 minutes.

Case No. 8 A.F.

BLOOD EXAMINATION

Date	2-23-27	3-5-27	3-16-27	3-30-27	4-14-27	4-24-27
Hgb.	84%	68%	68%	84%	85%	88%
R.B.C.	4,180,000	4,176,000	4,168,000	4,152,000	4,180,000	4,176,000
W.B.C.	8,600	8,400	8,000	7,800	8,400	8,200
Polys. Neut.	39%	37%	38%	37%	39%	37%
Eos.	2%	1%	3%	2%	1%	3%
Baso.		1%		1%		
Lymph. Small	57%	59%	56%	58%	54%	56%
Large	1%	2%				
At.		1%			1%	
Mones.	1%	3%	3%	5%	5%	4%

	CALCIUM	PHOSPHORUS	PRODUCT OF Ca. AND P.
2- 4-27	8.6	3.95	26.0
2-14-27	9.8	4.93	32.0
3- 1-27	8.0	4.28	25.5
3-14-27	7.9	3.57	28.2
3-28-27	9.9	4.7	46.5
4-11-27	9.5	4.9	46.5
4-25-27	9.9	5.5	54.4

ROENTGEN-RAY REPORT

The roentgen-ray studies showed progressive healing while the patient was having sunlight through the Corning glass window.

Premature infants and twins should receive prophylactic treatment. The same holds true, to a less degree, of negro and Italian babies living in the northern part of the United States. For premature infants and twins, the need of protection is so great that if oil is not tolerated, they should receive treatment by the other specific therapeutic agent—ultraviolet rays.

In hospital practice and individual cases where the diet is inadequate or there is a lack of sunshine, cod liver oil should be given as a routine prophylactic measure from October to May.

The influence on the disease by the use of

cod liver oil is rather graphically shown in an analysis by the cases during two periods at the Children's Hospital.

In 1923, when our special clinic in the Out-Patient Department of the Children's Hospital was organized for treating rickets, seventy-two cases were treated during the year. This clinic has been carried on since that time and, although the total number of babies treated in the Out-Patient Department has steadily increased, the "Rickets Clinic" has gradually decreased, so that during the past year only fifteen cases of rickets have been treated. Only the moderate and severe cases of rickets are treated in this clinic with ultraviolet light and cod liver oil. The mild cases of rickets are treated in the regular Medical Clinic with cod liver oil alone.

Orthopedic admissions to the ward for the postrachitic deformities and operations for these deformities for the years 1921 and 1926 were as follows:

Admissions

	No. of cases 1921	1926
Bow legs	52	20
Knock knees	18	6
Knock knees and bow legs	7	0
Cora vara	1	1

Operations

Bow legs:		
Manipulation	5	2
Reduction of tibia (opr.)	2	0
Osteoclasis	31	10
Osteotomy	20	6
Knock knees:		
Osteotomy	24	5

Table No. 10

Case No. 9 L. J. Colored female aged 22 months

RECORD OF SUNSHINE RECEIVED THROUGH VITAGLASS WINDOW 1-12-27

Week	Days of Sunshine	Total Exposure	Approximate time of day
1st 1-12-27 to 1-18-27	4	10 hours 45 mins.	11 a.m. to 2:45 p.m.
2nd 1-19-27 to 1-25-27	2	8 45	10:10 a.m. to 2:45 p.m.
3rd 1-26-27 to 2- 1-27	5	18 50	10:15 a.m. to 3:10 p.m.
4th 2- 2-27 to 2- 8-27	6	22 30	10:30 a.m. to 3:40 p.m.
5th 2- 9-27 to 2-15-27	6	12 10	12:30 p.m. to 3:40 p.m.
6th 2-16-27 to 2-22-27	2	7 15	10:40 a.m. to 3:30 p.m.
7th 2-23-27 to 3- 1-27	5	18 50	11 a.m. to 4 p.m.
8th 3- 2 -27 to 3- 8 -27	6	11 20	11:30 a.m. to 4 p.m.
9th 3- 9-27 to 3-15-27	3	10 25	12 n. to 4:30 p.m.
10th 3-16-27 to 3-22-27	4	8	1:30 p.m. to 3:30 p.m.
11th 3-23-27 to 3-29-27	3	7	1:30 p.m. to 4 p.m.
12th 3-30-27 to 4- 5-27	2	3 45	1:15 p.m. to 4 p.m.
13th 4- 6-27 to 4-11-27	5	10 15	1:30 p.m. to 4 p.m.

Total hours of sunshine through Vitaglass 147 hours 50 minutes.

Case No. 9 L. J.

BLOOD EXAMINATION

Date	12-20-26	12-27-26	1-10-27	1-25-27	2-10-27	3-4-27
Hgb.	62%	62%	62%	62%	62%	62%
R.B.C.	4,048,000	4,680,000	4,440,000	4,680,000	4,680,000	4,920,000
W.B.C.	8,600	8,400	6,400	26,000	8,600	8,000
Polys. Neut.	40%	38%	39%	51%	37%	39%
Eos.	1%	2%	2%	4%	3%	2%
Baso.	1%					
Lymph. Sm.	54%	58%	59%	40%	59%	58%
Large	1%				1%	
At.		1%			1%	
Monos.	3%	4%	4%	5%	5%	3%

	CALCIUM	PH SERUM	PRODUCT OF CA. AND P.
12- 7-26	7.55	3.42	25.8
1- 5-27	8.6	2.9	24.9
1-12-27	8.5	2.7	22.9
1-31-27	8.5	3.6	30.6
2-14-27	9.1	3.34	30.3
3- 1-27	8.3	2.75	22.8
3-14-27	9.5		
4-11-27	9.9	2.85	28.2

ROENTGEN-RAY REPORT

The roentgen-ray studies showed little or no evidence of healing during the patient's stay in front of the Vitaglass window.

During the past winter we have had considerable difficulty in finding a sufficient number of babies with severe rickets in the Out-Patient Department for our studies in the hospital ward, whereas, five years ago we had no difficulty whatever in finding plenty of babies with severe rickets. This striking decrease in the morbidity of rickets can be due only to widespread adoption of antirachitic measures which has resulted from Public Health propaganda and the recognition on the part of the general practitioner of the specificity of cod liver oil and sunshine.

There has been a marked diminution in the postrachitic deformities coming to the Orthopedic Department of the Children's Hospital during the past five years, which is good evidence that severe rickets is less common since there has been a more wide-spread policy of giving cod liver oil during the winter months. The

It is seldom necessary to use artificially produced ultraviolet rays for the prevention of rickets except in premature babies as rickets can be prevented in the majority of cases by cod liver oil and sunshine. In severe active rickets ultraviolet irradiation in combination with cod liver oil probably hasten the healing processes more than either ultraviolet rays or cod liver oil alone.

Our experiments make us believe that there are sufficient short rays in the sun's spectrum in Boston during the winter months to be of use in treating rickets as quartz and Corning glass windows transmit these rays in sufficient quantities to cure rickets.

Vitaglass, while it transmits light waves to the extreme limit of the sun's spectrum, in the two cases treated, did not transmit them in sufficient quantities to be of use in treating black babies during the winter months. It is possible,

however, had the windows been so situated as to give longer exposure to the sun's rays, our results would have been different.

One may look in the future to the use of ultraviolet transmitting windows in nurseries where the baby can bask in the sunshine in a warm room every sunny day during the winter months when rickets is so prevalent and do away with the necessity of giving cod liver oil as a prophylactic measure.

CHAIRMAN CRANDELL: Last month there was appointed through the Secretary a Nominating Committee to present nominations for the office of Chairman and Secretary of this Section for the coming year. That Committee was made up of Dr. Richard M. Smith, Chairman, Dr. John Lovett Morse, Dr. W. W. Howell, and Dr. J. Herbert Young.

Dr. Smith is on shipboard and I am going to ask Dr. Morse if he will present the report of the Nominating Committee.

DR. JOHN LOVETT MORSE: Mr. Chairman, your Committee nominates for Chairman Dr. Kenneth D. Blackfan, of Boston; and for Secretary Dr. Joseph Garland, of Boston.

Independently I should like to offer a motion that the Secretary be empowered to send the sympathy of the Society to Dr. Young, our Secretary, who is ill.

CHAIRMAN CRANDELL: I am sure that motion is seconded by everyone present and all in favor will please say "Aye"; opposed "No."

The motion was carried unanimously.

CHAIRMAN CRANDELL: It is a unanimous vote and I will ask the Secretary to transmit that vote to Dr. Young.

Now comes the question of the acceptance of the report of the Nominating Committee. What is the pleasure of the Section.

It was regularly moved and seconded that the Secretary be instructed to cast one ballot for the names as presented by the Nominating Committee.

CHAIRMAN CRANDELL: Before I put that question, are there any other nominations?

There were no other nominations. The motion was put to a vote and carried. The Secretary reported having cast the ballot and Chairman Crandell declared Dr. Kenneth D. Blackfan elected as Chairman and Dr. Joseph Garland elected as Secretary. (Applause.)

CHAIRMAN CRANDELL: The next paper is to be presented by Dr. John Lovett Morse. It is entitled "A Plea for Sanity in the Use of the Modern Methods for the Prevention and Treatment of Rickets."

A PLEA FOR SANITY IN THE USE OF THE MODERN METHODS FOR THE PREVENTION AND TREATMENT OF RICKETS

BY JOHN LOVETT MORSE, A.M., M.D.

RICKETS is no more common now than it was thirty years ago. At that time I studied four hundred consecutive infants under one year of age coming to the Out-Patient Department of the Infants' Hospital. This study was made in the summer and many babies in the first few weeks of life were included. Nevertheless, clinical, bony signs of rickets were found in 80%. These figures are about the same as those obtained now under similar conditions. I am unable to give any accurate figures, but I have a very strong impression from observation in private work, hospital wards and out-patient departments, that the average severity of rickets was steadily diminishing for some time before the introduction of cod liver oil and ultraviolet irradiation. Marked deformities of the chest and extremities were much less common than in the past, as were also malformations of the head and craniotabes.

Evidences of rickets can be found with the Roentgen ray in from 97% to 98% of young infants. In many instances, however, these are the only evidences of rickets. Under these circumstances the question at once arises as to whether these changes, which are supposed to be evidences of rickets, are not really normal, that is, physiologic. Under other circumstances, certainly, when a given condition is found in 97% or 98%, this condition is considered to be normal and its absence abnormal, instead of the reverse. Furthermore, these changes are more marked in rapidly growing and fat babies, that is, in those that are thriving the best. They are least marked in malnourished and atrophic babies, who are not doing well. These facts also suggest that these changes are not abnormal. They certainly show that they are not important. The fact that they develop in babies that have been taking cod liver oil from the time they were born, suggests that they may not be evidences of rickets or shows, as is pointed out later, that cod liver oil is not a preventive of rickets. It is also not improbable that slight clinical, bony changes, such as a slight rosary, may not really be a sign of disease. Certainly, when they are present alone, they do not mean disease of importance.

Nevertheless, much more is being made of rickets at present than in the past. This is due largely to the great amount of admirable work which has been done on the chemistry and etiology of rickets during recent years and to the discovery of the antirachitic factor and its relation to ultraviolet radiations. These discoveries constitute one of the triumphs of scientific investigation in medicine in recent years. Young

physicians have naturally been much impressed. They want to use the latest methods and, above all, to be "scientific." They must be in fashion. They also need practice. In consequence, they have made much of rickets. The lay press and mothers' clubs have helped them. They ought not to be blamed too severely, but, however, they have grossly exaggerated its importance. They have overlooked the facts that rickets is no more common now than in the past, that it is less severe than it used to be and that much of what is called rickets is probably not really rickets at all, but a physiologic change, and certainly not of importance. They have unnecessarily alarmed the Public and have made many mothers unnecessarily anxious. My policy used to be, when I found a slight rosary, a slight enlargement of the epiphyses at the wrists or a slight overgrowth of the cranial bosses, not to say anything about it and not to speak of the disease, rickets, because it seemed to me unjustifiable to alarm parents unnecessarily about a trivial condition which I knew would take care of itself. Nowadays, I always mention these abnormalities, if they are abnormalities, and tell the parents that their baby has rickets. I do this because I know that, if I do not, some young doctor or student friend of the family will mention them and the parents will feel that I have not noticed them and am a back number. I know that the young doctors think that anyway, but I prefer not to have the parents think so also. I do, however, say that, although I have to say the baby has rickets, the rickets which it has is of no importance whatever and there is no reason for them to be anxious. I give treatment or not, as seems to me advisable. The young physicians, and some old ones, however, emphasize the condition, make a great deal of it, frighten the parents and recommend all sorts of treatment. The question arises as to whether they are overenthusiastic and lacking in judgment or are deliberately exploiting the Public for their own advantage.

There is no doubt that cod liver oil, if it is active, that is, if it contains the antirachitic factor, cures rickets. There is also little doubt that it prevents the development of severe rickets. The studies of Eliot in New Haven and Wilson in New York show, however, that cod liver oil does not prevent, in the vast majority of cases, the development of mild, clinical rickets and has practically no effect on the development of roentgenologic rickets. The question at once arises whether the presence of the roentgenologic changes alone really signifies rickets and is not physiologic. It is evident, at any rate, that cod liver oil is not the universal panacea for rickets that it was supposed to be a few years ago. This being so, is it advisable to give all babies cod liver oil from the time they are born, regardless of how they are fed, their social status, how much sunlight they get and the time of the year? The work of McCollum and his collaborators shows that the antirachitic factor can pass through milk. The breast-fed babies of women taking a diet containing an abundance of this factor certainly need it less than artificially-fed babies. It seems hardly necessary for babies in the summer, if they get out of doors. Rickets of the grade which can be prevented by cod liver oil being uncommon, even in the winter, among the infants of the well-to-do, who get good medical care, it seems unnecessary to give them cod liver oil as a routine procedure. It is time enough when they show clinical evidences of the disease. The question arises as to whether there are any objections to giving it to them. There are probably no serious ones. It makes no difference to the doctor, even if it does to the mother, that the baby smells like a fish. This can be avoided, however, by giving the baby yolk of egg, which is an equally good antirachitic. There is some danger of upsetting the digestion with the fat of cod liver oil as with other fats, but this danger is not very serious. It may be well, however, for those who balk at the old-fashioned cream mixtures and feed all babies on whole milk modifications, to remember that one teaspoonful of cod liver oil contains as much fat as one ounce of gravity cream. It is always a good plan, moreover, not to give unnecessary treatment. The problem is different among the infants of the poor, who do not get constant medical supervision and never get as much sunlight as their more fortunate neighbors. They should be given cod liver oil as a routine. It must be remembered, in this connection, that rickets starts early, in the first few months of life, and that, therefore, the use of cod liver oil should be begun soon after birth.

The reason that cod liver oil, sunlight and artificial ultraviolet irradiation do good in rickets is now known. The ultraviolet rays, whether in sunlight or produced artificially, cause a chemical change in the cholesterol of the skin. This "activated" cholesterol in some way influences the metabolism of calcium and causes it to be laid down in the bones. The ultraviolet rays cause a similar chemical change in the phytosterol of plants. When sea plants, which have been activated by the sun's rays, are eaten by the cod, its fat is also activated. The activity of cod liver oil can be increased by exposing it to ultraviolet irradiation. Many other fats can be activated in the same way. So also can green vegetables and grains. Milk, even when dried, can be activated and will remain activated for some time. Irradiated cholesterol and phytosterol in all sorts of food may, therefore, be used for the prevention and cure of rickets. In some instances such foods may be preferable to cod liver oil and direct irradiation. In general, however, they should be looked on simply as "fads" or evidences of what can be done in medicine, rather than as practical aids in treat-

ment. It must also be remembered that irradiation may destroy Vitamin A and cause other undesirable chemical changes in food. Irradiation of nursing mothers and cows, although it has been suggested and, I think, done, seems rather extreme and unnecessary.

The lowest visible ultraviolet rays are about 390 millimicrons in length. The ultraviolet rays extend down to 185 millimicrons. In sunlight the waves are of high intensity from 397 to 340, of much lower intensity from 340 to 291, and there are none below 291. Rays longer than (313) 302 have no potency in rickets. The rays in the narrow band between (313) 302 and 291 are, therefore, the only active ones in sunlight. There is ample evidence that there are enough active rays in this narrow zone, although they are of relatively low intensity and constitute but one per cent. of the total solar radiation, to prevent and cure real rickets in this part of the world in the summer. It is known that these waves are diffusible and that out of doors these diffused waves are active. It is not necessary, therefore, although better, to have the sun shine directly on the baby. There is also some evidence to show that they are transmitted through clothing. As ordinary glass does not transmit waves below 340 (330), it is evident that it keeps out all those which are active in rickets. Sunlight transmitted through ordinary glass windows has, therefore, no antirachitic action. In exposing babies to direct sunlight in the summer in order to prevent or cure rickets, it must be remembered, however, that, in spite of their antirachitic action, the sun's rays can cause "sun-stroke" now, just as in the past, and that excessive heat in the summer still lowers a baby's resistance to disease, enfeebles its digestion and favors the development of the diarrheal diseases.

A very important question is whether there are enough of the antirachitic rays in sunlight in this climate to prevent and cure rickets in the winter. Unfortunately, no one knows just how much ultraviolet radiation is necessary at any time to accomplish this. Dr. Bovie is, I believe, trying to find out. All discussion of the subject must, therefore, be somewhat speculative. Certain things are known, however. In a clear atmosphere, 30% of sunlight is absorbed by the atmosphere when the sun is at the zenith and 75% when it is at the horizon. The sun being low in the winter, it is evident that a much greater proportion must be absorbed in the winter than in the summer. The hours of sunlight are much less in the winter than in the summer. According to the Weather Bureau the possible hours of sunshine in Boston during the three winter months are 284, 295 and 297. The average number of hours of sunshine during a long period of years was 136, 143 and 172 respectively, or 4.4 hours in December, 4.6 in January and 6 hours in February. In this connection it must be remembered, however, that, even in the summer, there are very few active rays in sunlight before 10 a. m. and after 2 p. m. There are also fewer short rays in the winter than in the summer. Furthermore, smoke, gases and dust in the air absorb these rays. There is much more smoke in the air and, in consequence, gases, in the winter than in the summer and, when there is no snow on the ground, as much or more dust. Moisture in the air absorbs these rays. In consequence, they are further limited in damp and melting weather. Reasoning on the basis of these facts, it seems improbable that there are enough active rays in sunlight in the winter to either prevent or cure rickets, even if a baby is directly exposed to them.

If there are, as Dr. Wyman's work seems to indicate, it would seem worth while to make use of them, if it can be done without incurring other dangers which will more than counterbalance any advantages which may be gained. If there are not, as is the general belief, it hardly seems advisable to take unnecessary risks for a very doubtful advantage. To make use of these rays in the winter, the babies must be exposed naked to them, either directly or transmitted through glass which allows them to pass. It is feasible, of course, under ideal conditions, to let a baby lie or play naked for a time each day in the sun in a warm room with the window open, provided the sun is shining. No one knows, however, how long this time must be. Furthermore, even in a warm room, there must be some danger that the baby will be chilled and hence made ill. It seems unreasonable, as well as dangerous, to put naked babies out of doors in the winter in this climate to avoid a disease, the importance of which is grossly exaggerated and which can be prevented, at any rate except in its mildest forms, by other means. Nevertheless, this is done by some enthusiasts. Cod liver oil, irradiated foods or even ultraviolet irradiation would seem more rational.

Provided that there are varieties of glass which will transmit these rays, and provided that these varieties are used, there is little or no objection to exposing babies to them naked in warm rooms. Such exposure may, however, result in chilling and illness, even when great care is taken. It is not practicable, however, except for the rich and well-to-do, unless the price of such glass comes down, and not worth while until it is proved that there is sufficient antirachitic power in the winter sunlight to do good and how much exposure to it is necessary to accomplish anything. At present, it seems inadvisable to go to the expense of putting in such windows or to recommend their general use. There can be no objection, of course, to putting them in for experimental purposes.

There is no doubt that the carbon arc and quartz mercury arc lamps are efficient producers

of ultraviolet radiations. The carbon arc lamp emits powerful rays between 397 and 300 millimicrons in length and many weak rays between 300 and 200. The quartz mercury arc lamp emits powerful rays between 397 and 300, strong between 300 and 230 and weak between 230 and 185. Both, therefore, emit many rays below (313) 302, which is the upper limit of the rays efficient in rickets. There is also no doubt that artificial irradiation with these lamps will not only cure rickets, but prevent its development. No objection can be raised to the use of artificial irradiation in the treatment of rickets. In fact, a cure can probably be obtained more quickly in this way than in any other. It must be remembered, however, that rickets can also be cured by the use of cod liver oil and sunlight, that cod liver oil is cheap and artificial irradiation expensive, that there is considerable danger of exposing infants, and thus causing other illnesses, when taking them to doctors' offices several times a week for treatment, that it is a great temptation to physicians, who have equipped their offices with lamps, not only to get their money back, but to get a premium on their investment, and that the instrument makers are anxious to let or sell to the Public as many machines as possible. Furthermore, in using artificial irradiation, it must also be remembered that pure radiologic rickets is very likely not rickets at all and certainly of no practical importance and that even slight clinical evidences of rickets in otherwise normal babies are of little more. The recommendation of artificial irradiation in cases of this sort, unless the treatment is given free, seems, therefore, to be taking advantage of the fears and gullibility of anxious mothers and little short of fraud.

As already stated, artificial ultraviolet irradiation will prevent the development of rickets. Should it be used universally for this purpose? Should all the rich babies have ultraviolet lamps in their nurseries? This would please the makers of lamps. Should all the rest who have the price, go to doctors' offices for regular treatments? This would be fine for those who have put in the lamps [redacted] are hoping for financial returns from them. Should the remainder go to the hospitals for treatment? This would swamp the hospitals. Such extreme measures seem hardly necessary when it is taken into consideration that much that is called rickets is very likely not rickets, that slight rickets is probably not important anyway, that the development of severe rickets can be prevented by the administration of cod liver oil and that, if rickets does develop, it can be easily cured. Let the rich have their lamps, if they want them, understanding, however, that there are potentialities for harm in them. Let the well-to-do go to doctors' offices for treatment, if they please, provided that they are told the real facts regarding rickets and are not exploited. Let the poor go to the hospitals, if the physicians in charge think they are justified in using money contributed by charitable persons or the public authorities for such a purpose.

It is apparently taken for granted that it is impossible to do any harm with artificial ultraviolet irradiation, even if it does no good. There is ample experimental evidence to show that the prolonged irradiation of cholesterol and of cod liver oil and foods which have acquired antirachitic powers, as the result of irradiation, diminishes and finally destroys these antirachitic powers. Furthermore, when cholesterol has been inactivated by artificial irradiation, it cannot be reactivated. It is evident, therefore, that too prolonged irradiation of babies may, instead of activating the cholesterol in the skin, not only inactivate it, but prevent it from being activated by sunlight. Unfortunately, there are at present no trustworthy data as to where the line between beneficial and harmful artificial ultraviolet irradiation is situated.

Further points to be remembered in this connection are that excessive ultraviolet irradiation kills certain small animals and that, under certain conditions, it may do serious harm to plants and seeds. Plants grown in the absence of light are "etiolated," that is "rachitic." This is because, in the absence of light, there is a lack of differentiation of tissues. An excess of light, that is, of ultraviolet rays, may, on the other hand, cause a too early differentiation of tissues and result in dwarfing. It is possible that the exposure of babies to an unusual amount of ultraviolet irradiation over a long period of time may cause an excessive deposition of calcium in the bones and interfere with or prevent normal growth of the bones in length.

There seems to be a general tendency among physicians to assume that, because ultraviolet irradiation does good in rickets, it will also do good in all disturbances of nutrition or prevent their development. It does good in rickets because it activates cholesterol and, in this way, favors the deposition of calcium in the bones. It cures spasmophilia because it increases the calcium content of the blood. It helps certain cases of asthma and of nervous exhaustion, in which the calcium content of the blood is low, for the same reason. There is no reason to suppose that other disturbances of nutrition are due to a disturbance of the metabolism of calcium. There is no justification for thinking, therefore, that because ultraviolet irradiation is useful in conditions in which there is a disturbance of the metabolism of calcium, it will do good in those in which there is not. Physicians who think at all why they use artificial ultraviolet irradiation for the improvement of nutrition, argue that it increases the basal metabolism. Sunlight undoubtedly does. How much of this action of sunlight is due to the ultraviolet rays which it contains, is questionable. Probably the red and

infra-red rays are much more influential. Furthermore, the patients are also exposed to the action of the wind and get fresh air at the same time. Physicians say that it benefits the blood. What is the matter with the blood in disturbances of nutrition? Is there anything the matter with it? If there is, is the trouble always the same? Can ultraviolet irradiation do any good, if there is? The ultraviolet rays penetrate only 0.1 to 1.0 millimeters. The shorter, and presumably the more active, rays, do not even reach the basal layer of the epidermis. It is evident that they cannot have much direct action on the blood. Moreover, the results of studies of the action of the ultraviolet rays in anemia, the condition most likely to be affected by ultraviolet irradiation, are conflicting and inconclusive, and, in the main, show that they have no action. Physicians say that ultraviolet irradiation will kill bacteria. They will, under the most favorable conditions. Not even the most enthusiastic exponent of ultraviolet irradiation would go so far as to claim, however, that all disturbances of nutrition are due to bacteria in the epidermis, which is the only place where the ultraviolet rays could reach them. They claim that, by its action on the nerve endings in the skin, ultraviolet irradiation may reflexly affect the depths!! Maybe it can. I do not know, neither do they. As an argument, however, it is "bunk." They also say that adults feel better after a treatment. I have no doubt they do. Anyone will feel better, if he undresses, lies down quietly, first on his back and then on his abdomen, in a warm room for half an hour to an hour, especially if his mind is at rest and he has confidence, however misplaced it may be, that something marvelous is being done for him. There being no scientific evidence to show that ultraviolet irradiation does good in either preventing or curing general disturbances of the nutrition, it seems to me that it is unjustifiable either to recommend or use it for these conditions. That people like it, that it increases a doctor's practice and his income, and that it is the fashion, are not sufficient arguments to justify its general use. Before it is employed generally in these conditions, its action should be thoroughly and scientifically investigated in institutions and by men who are competent and unprejudiced. Until this is done, it is well for us to remember the words of Pope's familiar couplet—

> "Be not the first by whom the new is tried,
> Nor yet the last to lay the old aside."

(Applause.)

CHAIRMAN CRANDELL: Several gentlemen have been invited to discuss these papers that have been presented by Dr. Gamble, Dr. Wyman, and Dr. Morse, and first upon the list is the name of Dr. Kenneth B. Blackfan, whom I should like to introduce to you.

DR. KENNETH B. BLACKFAN (Boston): Mr. Chairman, Ladies and Gentlemen: Sir William Osler once said it is of use from time to time to take stock, so to speak, and find out our knowledge of a particular disease. When we recall the tremendous activity in the investigation of rickets which has been going on for the past few years, it would seem that this was an appropriate time to take stock of what we know about the cause and the treatment of rickets. I feel that we have been particularly fortunate this morning in having had the various aspects of this disease presented to us by such authorities as are gathered here.

Finality regarding the cause of rickets has not been reached and yet I am sure that even Dr. Morse will not commit me to a psychopathic institute if I make a few general remarks concerning the situation of this form of rickets, acute infantile rickets, as has been presented by himself, Dr. Wyman, and Dr. Gamble. The cause of rickets is not known, yet certainly diet, environment, and lack of sunshine are strong predisposing factors towards its development and also it is known that its prevention and its cure are facilitated by the use of cod liver oil and sunshine.

The speakers have been discussing this morning acute infantile rickets, a disease with which you are all familiar, and yet I feel that a distinction should be made between bony deformities which are due to other factors; for instance, in certain forms of renal diseases there are deformities of the bones and the roentgenological changes are identical as well as the lowering of the calcium, or the disturbance of the calcium product, also in certain forms of chronic intestinal indigestion, particularly the form generally spoken of, probably improperly, as celiac disease, roentgenological changes, and calcium phosphate disturbances, may be found simulating infantile rickets.

In the children's clinic Dr. Hoeffel has collected a relatively large number of children with cinical aspects of rickets, roentgenological changes and changes in the blood factors. The cause of this latter group has not as yet been determined, but in these conditions which are clinically identical, and by the laboratory data are similar to acute infantile rickets, our attempts to cure this disease by the use of cod liver oil and sunshine are disappointing.

There is an opportunity for skepticism regarding the effects of cod liver oil and sunshine in certain conditions which have been commonly supposed to be pathognomonic of infantile rickets. I refer to craniotabes. Dr. Sisson has found at the Lying-in Hospital in Boston that in premature infants showing marked and extensive craniotabes, the x-ray changes characteristic of acute infantile rickets are not present nor is there a disturbance of calcium and phosphorus products of the blood. This

undoubtedly may be considered, as Dr. Morse has suggested, as physiological rickets, if you please, but certainly is not the type of rickets which is prevented or cured by the use of cod liver oil or sunshine.

Regarding Dr. Wyman's studies, I think they are of fundamental importance and they may prove of value in the keeping up of the health of the young infant, because it is obvious to all of us that infants are in better state of nutrition and are less susceptible to infections if they are bronzed or tanned and if they have access to the sun's rays. These contributions I feel are of great importance.

In conclusion I wish to join with Dr. Morse in protesting against the promiscuous use of commercial ultraviolet rays. These therapeutic rays should be used only for those diseases for which it has been shown by critical evidence that they are effective and the number of these diseases is relatively small at the present time; that is, in rickets, in tetany and in certain forms of tuberculosis; until further information is obtained, they should not be used as a means of treating any disease just because it is a popular form of treatment.

(Applause.)

Dr. Fritz B. Talbot (Boston): Mr. Chairman, these papers have been extraordinarily interesting and timely. The one thing that impressed me during the discussion was the lack of emphasis on the fact that rickets is not a disease alone of the bones, but it is a general disease, a disease of the whole body and the muscles and muscular system and other systems also are not normal during the acute stage of rickets.

I think in any consideration of the subject that should be kept in mind and the possible late results that may follow as the results of other parts of the body, should be taken into consideration in summarizing and balancing the subject.

I agree with the speakers in nearly all that they said. It seems to me that they have presented the subject in a very comprehensive manner. I should like to reëmphasize the fact that the late results of rickets are disappearing from the community. I took opportunity to ask men from various parts of the country about their experience with rickets and the following places say that they do not see the late deformity of rickets nowadays in the same numbers as they used to see them: Baltimore, New York, Chicago, University of Virginia, Kansas City, Montreal, and Minneapolis. I have no doubt that the same things would apply to other parts of the country.

Human nature hasn't changed any from the time that I started assisting Dr. Morse. I remember very well that there were confreres who at that time, too, were alarming patients with the diagnosis of rickets and Dr. Morse's remarks apply to fifteen years ago to the same extent that they apply today.

I think also that his emphasis of the unnecessary administration of cod liver oil in private practice is a very important point.

In regard to the presence of the ultraviolet rays, they are present in Boston, as the speakers have emphasized, and they have been studied very carefully in Toronto, in surroundings and latitude the same as Boston, and they find that there are ultraviolet rays present and that they are active during the winter months.

A rather important finding that Tisdale reported this year is that they are rather more strong not directly in the sunlight itself, so that removes the necessity of putting a baby directly in the sunlight and if that is so, then we can please all the grandmothers and they won't have to worry about the baby's eyes when we are giving the ultraviolet light.

The expensive glasses that are used and recommended for windows, of course, make it impossible for many people to make use of this method of prevention, and I should like to ask Dr. Wyman whether he can enlighten us any on the explosive celo glass which is supposed to be useful in bringing up chickens. Perhaps our chickens may be able to take advantage of that.

To summarize, this whole discussion brings up again what I think is the most important step in modern medicine today. It emphasizes how the medical public's point of view is changing, how we are all looking more and more to preventive medicine, and if some of us look a little bit too high and talk a little bit too much, perhaps it is excusable if we get the results which remove the deformities which in the past ruined the lives of at least a small number of the population.

Dr. Orville R. Chadwell (Jamaica Plain): Roosevelt said that there was a lunatic fringe to every great movement.

After Dr. Morse's paper, I am very loathe to admit that it has been my custom in general to prescribe cod liver oil for most of my babies. In our child hygiene clinics controlled by the Board of Health in Boston we have made it almost a routine. To me it hasn't seemed altogether safe to try to discriminate in the choice of the babies who need this sort of medicine.

Dr. Wyman brought out one interesting point to me and that is the rarity with which we see definite rickets in our clinics. It is rather hard now to show medical students any number of cases during their courses in pediatrics. It is still rarer to show them tetany.

I believe that inasmuch as our clinical material for study in our schools is drawn from the same class of children here in Boston who would naturally attend our child hygiene clinics, that the absence of these deformities in our out-patient clinics may have some relation to the use

of cod liver oil in general, as I think most of the men do prescribe it.

(Applause.)

DR. MARTIN J. ENGLISH (Boston): I have little to say on the very complete and exhaustive papers and discussions that you have had from Dr. Gamble, Dr. Wyman, Dr. Morse and the men who have discussed the papers just previously.

I am very fortunate to be one of the disciples, if I might say so, of Dr. Morse. I have seen the medical schools go through a number of changes and as a result of possibly many of those great changes, I have been able to follow the conservative way, usually, in pediatries.

I think the great result of an exhaustive discourse like this produces one thing: it has been a tremendous factor in educating the public, so much so that the municipalities have taken up this great subject and that, I think, has been a great factor in the marked decrease in the incidence of many of our diseases. Take the question of scurvy—I think that is another disease in addition to rickets which is becoming extinct in our children. In City Hospital I think we have seen only two or three cases in the last few years. In the old days of the Children's Hospital, the late Dr. Rotch could show half a dozen cases, so that is a question of education. The mothers are being taught methods of preventive pediatrics through community health organizations, and by social service workers. They are getting ideas on how to take care of their babies. The newspapers and periodicals also help in this. Excerpts from discourses such as this are printed in the daily newspapers and there is no question but many of the people absorb what they read, the results of our experimental work in the laboratories, the results of work done in the clinical side in the hospitals and the work that is being done by organizations like the social service and community health organizations, private or public.

I think one sees in a big out-patient clinic that the mothers know that cod liver oil should be given early. They have an idea or they learn from discussions or what they read in the newspapers that cod liver oil should be given early because the disease has its inception in the first three or four months and they see the results after that time. Consequently, they know it should be given at the end of the first or second week or at least towards the end of the first month.

They know that as they know that orange juice or other citric fruit juices are a direct specific against scurvy, so I think the public as a whole is distinctly benefited by such discussion as we have heard here today and where it is transmitted to the newspapers and through the different periodicals, there is a distinct advantage and a distinct help to everyone in general.

I also have noticed in our big clinic at the City Hospital that the incidence of rickets is tremendously low as compared with the number of cases we used to see years ago, not only as to numbers, but the mild tie-up of rickets that we see today.

Of course, in pediatrics, as in other branches of medicine, the chief thing is diagnosis; that is, what is the cause? What is the source of the individual clinical or pathological condition? There is no direct distinct causative factor for rickets that we know today. It may be hereditary. There are some men who are writing about the question whether or not rickets isn't an hereditary or congenital thing. That would have a tendency to bear out Dr. Morse's statistics.

In a great many new-born babies you find ricketic tendencies or slight manifestations that look like rickets that you see usually in the second or third, or fourth or fifth month, not knowing the exact causation. Consequently, we still have to remain in a state of empiricism. Consequently we use or apply therapeutic measures to remove certain clinical or pathological disorders. Consequently we use cod liver oil and put the baby in the fresh air.

It is absolutely essential that the food should be properly balanced. There is no question but what a child that has plenty of fresh air and sunshine, has cod liver oil and has food that has proper carbohydrate qualities and salts can escape rickets.

Of course, fresh air and sunshine and irradiation by the artificial lights do not protect the baby to the same extent as if the food were properly balanced. Consequently, I think the first thing to do is to get the baby on a sound rational food basis and use these other things as helps to improve something that may be absent.

I think another important thing that Dr. Wyman has stressed very much is the care of the premature baby. There is no question but what we are saving many more babies than we were a year ago. First, of course, we have breast feeding. I think it was Dr. Talbot who was the originator of the wet nurse directory. A great many are using it now. We are using it at City Hospital and we feel that the morbidity and low mortality in premature babies is largely due to that particular fact that here in Boston we can get breast milk.

There is no question but that irradiation or exposure to sunlight or to the quartz lamp helps many of those premature babies and I should like to emphasize the fact that with breast milk and with radiation through the quartz light or Corning light, we are still going to save a great many of the premature babies.

I feel, too, as Dr. Morse does that the question of cod liver oil doesn't have to rest on giving it to the child, every individual child, that is. You have to pick out and select the cases. It is the

same with the radiation by the ultraviolet ray. There are many babies who do not need it. It does no harm, in my experience, to give them cod liver oil provided it does not disturb their digestion.

The point that Dr. Morse brought out, I think, most of our food indigestive babies are usually associated with too high fats and if the baby is getting extra fat by cod liver oil, you are more likely to get indigestion from that.

(Applause.)

Dr. Edmund B. FitzGerald (Wollaston): I think when Francis Glisson first described Rickets in 1650 he did not realize what future discussion he should cause. Recently I saw a pediatric text book, written in 1800, discussing rickets in which it was spoken of as a recent disease—great emphasis being laid on proper food and sunlight; so there doesn't seem to be very much new in the treatment of rickets.

During the last four years I have been supervising the child welfare clinics in an adjacent city and, although my examinations were very superficial and not at all to be compared with those made, since 1890, by Dr. Morse, I have certain figures that might interest you.

There were a total of 1660 examinations made in four years. In this number I made a diagnosis of active rickets 163 times. I made a diagnosis of "rachitic deformities"—a term which I used for an old rickets, presumably healed, 62 times. My assistants made a diagnosis of acute rickets in 62 cases and diagnosed rachitic deformities in 34 cases. That made a total of 225 cases of acute rickets among 1660 babies—about one eighth—and 96 cases of rachitic deformities.

Some time ago an ignorant and irate father came to my office considerably excited about a diagnosis of "rickets" that I had made and blurted out the question "What is them rickets anyhow?" From that time on I began to speak less about rickets to my patients.

Cranio-tabes can be elicited in almost any young baby providing you press hard enough and it is rather impossible at the present time to arrange any apparatus by which the pressure can be read off in terms of pounds and ounces.

As far as cod liver oil is concerned I feel that it is good insurance; that it should be given to every bottlefed baby in the winter time. Not too much should be said about rickets when you are prescribing cod liver oil and I rarely mention rickets to the mother, at the present time, unless I feel, as Dr. Morse felt, that somebody else is going to see the baby, or unless I feel that the disease is so obvious that the mother herself suspects it.

It has sometimes been obvious that we get better results in out-patient cases, where we have a careful follow up system, than in private cases where we may not have home conditions and diet under such careful control.

I have frequently noticed that babies with a fat indigestion which wouldn't permit them to digest a formula containing one percent butter fat will assimilate a fairly large dose of cod liver oil with no apparent signs of indigestion. Cod liver oil may be given to older children in a concentrated form which I believe has been accepted by the Council of Pharmacy and Chemistry.

I never give cod liver oil to breast fed babies because if rickets does develop in a breast fed baby it is usually so slight that it is a disease of very short duration. I always give it to twins or premature babies and start very soon after birth.

There has been some interesting work done by Dr. Maynard Ladd in preventing rickets. Five groups of babies were studied at the Boston Dispensary. The first group were given pasteurized milk for a definite period. Their percentage gain was very low: 1.7%. Another group had pasteurized milk plus orange juice. Their percentage gain was 7.9%. A third group had pasteurized milk, cod liver oil and orange juice. Their percentage gain was 9.5%. A fourth group got certified milk alone and their percentage gain was 14%. A fifth group got certified milk, cod liver oil and orange juice. This was a group of only five cases, so it is hardly fair to use as a basis of opinion, but their percentage gain was 7.2%.

Every three months X-ray examinations of the wrists were made by Dr. Friedman, Chief of the X-ray Department of the Boston Dispensary.

In the certified milk series no cases of clinical rickets were discovered and only three showed a mild self-healing X-ray rickets.

May it not be that a certain improvement in the milk supply in the last few years along with considerable improvement in housing conditions has had something to do with this marked diminution in rickets which has been noted by the other speakers?

Dr. A. C. Eastman (Springfield): It seems to me that this discussion of these papers has brought out one factor rather definitely, emphasizing that during the last twenty-five or thirty years probably the specialty of pediatrics has done as much as any specialty in scientific progress, and, as Dr. Morse has told us in his paper and hinted quite strongly, as it is a specialty, we have to be more careful perhaps to keep our feet on the ground. So many of these new problems arise! New results of experiments give us new ideas and very easily develop fads.

I have been very much interested in Dr. Morse's paper from that point of view.

I also would like to leave the question whether or not the noticeable decrease in rickets during the last few years, which has been spoken of by several of the men, may not possibly have some-

thing to do with the emphasis that has been placed upon breast feeding; whether or not the increase in breast feeding and the popularity of breast feeding among the average young mothers of the present day perhaps hasn't left its mark already in the diminution of some of these conditions which we may be inclined to think are entirely due to the use of cod liver oil or ultraviolet light. (Applause.)

DR. EUGENE C. PECK (New London, Conn.): There are two practical points about sources of ultraviolet light which I should like to mention. One is in regard to Celo glass. I find that a tuberculous poultry raiser in New London introduced it in New London and it has been used for nearly two years successfully in various heliotherapy cases.

The other point is in regard to carbon arcs. Those of you who take motion pictures realize that there are carbon arcs available on the market at present. A pair of carbon arcs is used in the lamp for taking motion pictures indoors. One of those arcs is similar to those used by Sir Henry Gauvain and others in England for the treatment of tuberculosis and possibly also of rickets. It will tan a baby. Those arcs are not protected by such exclusive patents as the mercury quartz lamps and are quite cheap, in fact, cost less than $100. They are a good source of heat and I think it is possible that they might be used as an open fireplace was sometimes used for undressing and dressing the baby and handling him. It would be well worth your while to look over those lamps for sale by the larger photographic houses dealing with amateur movie apparatus.

DR. F. H. ALLEN (Holyoke): I should like to ask Dr. Wyman a question. It seems to me if we are going to give cod liver oil, we should give a cod liver oil that is potent. I find often times mothers are getting cod liver oil not physiologically tested, or alcoholic extracts of cod liver oil. If we are going to use it at all, we should get a potent cod liver oil,—one physiologically tested on white mice. I should like to ask Dr. Wyman what he uses.

DR. E. W. BARRON (Malden): In regard to that I should like to make a remark. During my service in the Boston Dispensary in March, April, and May, I had a window of celo glass, and using it on three babies only, I was able to produce a certain amount of tan in that time. I thought I would report that.

DR. FRANCIS P. DENNY (Brookline): I think in the discussion of the use of radiation, either from the direct sunlight or from the lamps, we ought to feel that the benefit is not merely in the curing of rickets, although rickets is the subject under discussion. I think it has been definitely proven in the treatment of children with glandular tuberculosis that it is a benefit, and perhaps it acts in ways that we do not understand.

Nothing has helped in the treatment of tuberculosis except what improves the general health and I think we must feel that in exposing children and babies to radiation, we are doing something else than curing rickets. We weren't intended to live indoors and wear thick opaque clothing all our lives. (Applause.)

DR. J. W. SHERMAN: My remarks are not particularly scientific, but about a year ago this time I had occasion to spend a vacation on the Gaspard Peninsula at the mouth of the St. Lawrence River and I was interested in watching the fishermen working on the elementary production of cod liver oil. The fishermen come in with the boat loaded with codfish which they proceed to open on the shore, throwing the livers into dirty, filthy buckets, where they stay exposed to the sun, to quantities of flies, and to the odors of decaying fish, the waste product being thrown on the beach, and not until the boatload of cod had been dissected by one or two men were those cod livers removed to a more sanitary place.

I looked about to see if there was any evidence of ice, but I understood the cod livers were placed in a cool place until packed in barrels.

The question that comes to my mind is, Is the future of the human race dependent on such an unnatural and uncleanly source of food? Also, has the medical profession given attention enough to the other food factors which contain the vitamin constituents so necessary?

In a few years from now I wonder if someone with the courage Dr. Morse has had in attacking the electro-therapy will realize the lack of indication in the other things besides cod liver oil as a source of food for babies.

DR. JACOB LUFTIG (Boston): I wish to discuss the dosages of various remedies for rickets that were brought up today. In my opinion, if you are going to use cod liver oil, I believe that very small doses are all that you need to get the remedial effects of cod liver oil. In the past they have used three, four, and five teaspoonsful a day. It has been my custom in private practice and in the clinics to use as low as five drops of cod liver oil in each bottle in a modified-feeding case, and in the breast-fed babies to use about thirty drops a day.

You see, that is very small dosage compared to what some of the men are using and what has been used in the past and I find that I am getting equally as good results with possible avoidance of the fat indigestion that has been spoken of.

I rarely use the cod liver oil beyond five months and possibly beyond four months in some cases. I switch to egg yolk

as rapidly as I think the baby can stand it. Egg yolk is a very valuable anti-ricketic agent and should be used as soon as it can be taken instead of the cod liver oil. I recommend its use as low as four months and in many of the bottle-fed babies as low as five months, either hard-boiled or preferably soft-boiled two-minute yolk mixed with the feedings.

Concerning ultraviolet radiation if you are going to use ultraviolet at all, and it should be used only in severe cases of rickets, the tendency should be to use greater distances in inches away from the lamp; I believe that forty- to fifty-inch distances, and even greater if you can get it, are more valuable than the thirty- and twenty-five-inch distances. In other words, I try to avoid the hyperemia which is not at all necessary in ultraviolet radiation.

DR. E. P. BAGG (Holyoke): Before you close, it seems timely to raise the possibly hypothetical question whether or not too promiscuous doses of cod liver oil do not result in neurolytic action, judging from the work of Dr. Miner. At any rate, I know that it does promote an erection and possibly there is an element of anemia which may be overlooked.

CHAIRMAN CRANDELL: I will ask Dr. Gamble if he has anything he wishes to add.

DR. GAMBLE: I have nothing to add.

CHAIRMAN CRANDELL: Dr. Wyman!

DR. E. T. WYMAN: Celo glass is very transparent to ultraviolet rays. The reason we have not used it is that it is thin and a good deal of cold will come through it, it collects dust and is not easily washed. It is necessary when using these ultraviolet-ray-transmitting glasses, to keep them very clean. They should be washed every morning so that dust or soot will not collect on them.

In regard to cod liver oil, I think it is very important to get a clean cod liver oil. At the present time there are on the market a number of cod liver oils that have been tested biologically and that are produced in a cleanly way. It is worth while to take a trip to Gloucester to see the modern cleanly method the Patch Company use in producing their cod liver oil.

DR. J. L. MORSE: I think everybody knows I like to disagree with other people. I dislike to hear "tetany" used for "spasmophilia." Tetany is the name of a symptom that occurs in spasmophilia. I don't see why we mix up spasmophilia and rickets the way we do. It seems to me it certainly leads to clarity in thinking if we think of rickets and then think of rickets with spasmophilia, not of two different kinds of rickets.

I want to emphasize the danger of chilling babies in the winter. The physical laws governing the relation of surface area to bulk and loss of heat are the same now as they always were. The loss of heat by radiation is just the same as it always was. Dr. Wyman's babies were kept in a temperature of 80 degrees Fahrenheit, the naked ones, and didn't catch cold; nevertheless, the neutral point is 86 degrees and they must have been losing heat all the time and having to take extra calories so they had it to lose.

I don't agree that tanned children are always the healthy ones. We see a child at the seashore or coming back from vacation and he is all tanned up and we think he is very strong and resistant, but my experience has been that children who come back from the mountains and the country, not so much tanned, have better resistance than the children all tanned up from the seashore.

And one more thing in relation to what Dr. Denny said—we musn't confuse sunlight with ultraviolet radiation. There are many other than ultraviolet rays in sunshine. There are the red and the infrared outside the light. Furthermore, when babies are exposed to sunlight, they are out of doors and have the tonic action of the wind and they are breathing fresh air. There is a lot of difference between that and ultraviolet rays from an artificial source.

CHAIRMAN CRANDELL: I want to express to the readers and to those who contributed to the discussion of the papers the thanks of this Section.

This concludes the meeting of this Section and a motion to adjourn is now in order.

Upon motion regularly moved and seconded, it was voted to adjourn. The meeting adjourned at eleven-thirty o'clock.

STANDARDIZED MEDICAL SUPPLIES ADVOCATED ON AMERICAN VESSELS

In an endeavor to bring about standardization of medical supplies carried on American vessels the Bureau of Public Health Service, at the request of the Bureau of Navigation, Department of Commerce, has prepared and made public three standard lists of supplies designed to meet the needs of three typical sizes of vessels. The situation which led to this action by the Public Health Service was explained in a statement prepared by Dr. F. C. Smith, Assistant Surgeon General and Chief of the Marine Hospital Division, Public Health Service, which was made public at the Bureau on August 20.

The necessity for having aboard suitable supplies to carry out medical directions obtained by radio is an incentive. Probably the strongest incentive, however, is the fact that all ships' officers licensed by the Steamboat Inspection Service are now required to be trained in first aid. This involves a knowledge of many simple remedies which these officers now desire to have aboard ships.—*United States Daily.*

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 13361

THREE WEEKS' NOCTURNAL WHEEZING AND DYSPNEA

MEDICAL DEPARTMENT

An English chauffeur forty-two years old entered February 10 complaining of dyspnea.

For the past five years he had been somewhat slow in regaining breath after exercise, although twenty-one months before admission he won a 200 yard dash over fifty competitors with no bad results. Thirteen months before admission he had his first real dyspnea on carrying a bag of wood up four flights of stairs. After this he had other attacks of dyspnea lasting days at a time, accompanied by a sense of fullness and tenderness in the epigastrium. All the symptoms cleared up with rest, but sometimes this required several days. Occasionally on deep breathing he had short sharp epigastric pain. Nine months before admission the dyspnea became very severe and the sense of fullness was constant. His feet were somewhat swollen. After rest in bed for two weeks with digitalis he felt perfectly well, but had to limit his activities a little. He continued digitalis for three months and was practically symptom-free for three months after stopping it. Three months before admission he again had dyspnea and a sense of fullness, relieved by digitalis and rest. After another period of feeling well he had a severe chest cold five weeks before admission, with mild fever and chills, severe cough with a good deal ("a pint") of greenish and occasionally blood streaked sputum, rare short sharp non-radiating pain beneath the upper sternum, and profuse night sweats nearly every night. Since the onset he had not been able to work. The dyspnea and sense of fullness increased. At times on taking a deep breath he had pain in the side of the upper right chest. For three weeks he had had severe asthmatic attacks every night. For two weeks he had had to sit up every night because of coughing, dyspnea and wheezing. On two occasions he was relieved for a few hours by "abdominal injections" given by his physician. A week before admission he began to feel better except for marked weakness and loss of appetite, which he had had since the onset of the "cold". Under large doses of digitalis during the past week the cough practically disappeared and the dyspnea and sense of fullness became less marked. For a week he had slept very little. His feet had been swollen. For three days he had taken no digitalis. Two days before admission he vomited.

The little he knew of his family history is not significant except that his wife had had two miscarriages. Twenty-four years before admission he had gonorrhea. Otherwise he had always been strong and healthy except for three attacks of asthma, the first twenty years before admission, apparently not related to season or food. He had frequent headaches. Within twelve years he had had an inguinal hernia repaired. His wife had noticed a yellow tint to his skin at times. He thought at these times that his stools were clay colored.

Clinical examination showed a fairly well developed and nourished man, easily dyspneic, with very cyanotic lips and slightly cyanotic finger tips. Teeth poor. Very marked pyorrhea. The whole right chest in front showed a low harsh musical inspiratory and slight expiratory wheeze. A few dry râles below the angles of the scapulae after cough. Apex impulse of the heart heaving, seen 12 and felt 13 centimeters to the left of midsternum. Left border of dullness 13 centimeters, 4.5 centimeters outside the midclavicular line. Supracardiac dullness 4 centimeters. Marked apical thrill. Slow fibrillation. A rough systolic murmur heard all over, best at the apex. Second sound not heard. No definite diastolic heard. First sound loud at the apex. Pulses normal. Artery walls thickened. Blood pressure 148/98 to 140/103. An electrocardiogram February 11 showed auricular fibrillation, rate 90, right axis deviation with slurring QRS, —probable intraventricular block. Palpation of the abdomen was difficult in the Gatch bed. The liver was not definitely felt, but was apparently enlarged to both palpation and percussion, especially toward the midline, and very slightly tender. No definite shifting dullness or fluid wave. Very slight pitting edema of the lower legs and sacrum, not of the ankles. Pupils normal. Fundi showed peculiar pigmentation, possibly congenital. Knee-jerks sluggish. Ankle-jerks apparently normal.

Urine 10 to 38 ounces, specific gravity 1.020 to 1.030, a very slight trace to a large trace of albumin at all of nine examinations. A two-hourly test February 12 showed a large trace in all of five specimens, specific gravity 1.030 in four, 1.027 in one; no blood or pus. Renal function 50 per cent. Blood: 18,400 to 28,100 leucocytes, 80 to 81 per cent. polynuclears, hemoglobin 80 per cent., reds 5,120,000 to 5,350,000. Slight achromia. Wassermann negative. Vital capacity 850 cubic centimeters. Sputum not foul. No tubercle bacilli. Stool negative.

X-ray showed the heart shadow increased in size. The lung markings were prominent.

Temperature 97° by mouth to 101.6° by rectum. Pulse 70 to 129. Respirations 10 (morphia) to 40.

Orders. February 10. Fluids ad libitum. Morphia 1/6 grain; repeat once if very restless. February 14. Adrenalin 10 minims. Nitroglycerin 1/100 grain. Morphia 1/6 grain s.c. if not relieved. 1/2 cubic centimeter cardiazol at 8.20 a. m. and 8.40 a. m. 15 grains caffein benzoate s.c. 1 cubic centimeter digalen s.c. every four hours unless nauseated. Milk p.r.n. Morphia 1/8 grain every four hours beginning at 6 p. m. February 15. Soft solid diet. Morphia 1/8 grain s.c. every four hours by the clock unless respirations get below 15. Digitalis leaves 3 grains every four hours unless toxic beginning at 6 p. m. to-night in place of the digalen. February 16. Morphia 1/8 grain s.c., digitalis three grains, both given at 2.20 and 6.20 a. m. February 17. For collapse give the following in order with five minute intervals unless the patient's condition is extreme: (1) Adrenalin 10 minims. (2) Cardiazol 1 ampule intramuscularly. (3) Caffein sodium benzoate or salicylate 15 grains intramuscularly. (4) Morphia 1/6 grain s.c.

Examination of the heart February 11 showed the right border 3 centimeters to the right, the supracardiac dullness 6 centimeters. The apex impulse was diffuse, slapping, and absolutely irregular. There was a very loud, rather high-pitched harsh systolic murmur going very far out into the axilla and into the back and leading up to a weak second sound. Diastole was absolutely clear in all positions. At the aortic area there was a lower pitched, less loud blowing systolic murmur. The pulmonic second sound was accentuated, and the aortic systolic appeared there as well, less loud. Over the apex there was a systolic thrill. February 10 the non-protein nitrogen was 48. A blood culture February 12 showed staphylococcus albus in one flask, mould in the other.

The morning of February 14 the patient's condition suddenly became alarming. He grew very cyanotic and had rapid shallow breathing without expiratory wheezing. The radial pulse could not be obtained. The heart sounds were very rapid (over 130), absolutely irregular, rather poor in quality. He was semicomatose and muttered that he was dying. No râles could be heard in the front of his chest. The back was not examined. His feet and legs showed slightly more pitting edema than at admission. He was given 10 minims of adrenalin, an ampule and a half of cardiazol, an ampule of digalen and 15 grains of caffein sodium benzoate. In the course of an hour his condition seemed slightly improved.

That afternoon an arterial puncture was done and the oxygen content of the blood found to be 85 per cent. (The lower normal limit is considered to be 95 per cent.) At five o'clock the house officer was amazed to find the heart absolutely *regular*, the rate 110 to 115, the sounds of fair quality. There was no rub. The left border to percussion and the apex impulse were as at admission. The patient's general condition was much better. He was talking and perfectly clear mentally. The cyanosis seemed less, the breathing was deeper though still quite rapid, and there were no râles. The edema was the same. The spleen was not palpable in the orthopneic position. No petechiae were seen. The temperature was 101°, the leucocyte count about 20,000.

An electrocardiogram February 15 showed auricular flutter, 2:1 block, auricular rate 240, ventricular 120, right axis deviation with slurring Q R S,—probable intraventricular block. The non-protein nitrogen was 52. A consultant wrote: "Mitral systolic and diastolic murmurs heard, the latter only with the bell at the apex. . . . Why the fibrillation should change to flutter I do not know—perhaps the emergency medication (adrenalin etc.) may have been responsible yesterday. His prognosis is unfavorable unless the ventricular rate can be controlled and the infection cleared up. Death from congestive failure is likely. Digitalization again now to increase the heart block and perhaps to bring back auricular fibrillation seems indicated. If this proves unsuccessful quinidine may restore normal rhythm, though there is some risk in its use and improbability of satisfactory action. Vagal pressure fails to increase the grade of block."

February 18 the patient was doing badly,—dyspneic, orthopneic, cyanotic and edematous. He was toxic from digitalis. The pulse was still about 120, the temperature about 101° and the leucocyte count 25,000 without obvious cause. A third electrocardiogram showed auricular fibrillation, rate 120, right axis as before, with wider Q R S,—intraventricular block, left type. February 19 he died.

Discussion

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

This amount of fullness and tenderness suggests a congested liver but nothing worse.

I do not know what the short sharp epigastric pain on breathing means. It has no connection with any disease that I can name.

This is a good example of failure of compensation coming out all right through rest and digitalis.

Night sweats have traditionally been associated with tuberculosis, but if we look for them we find them in a great many other conditions. I think they mean nothing except fever. Why some febrile patients have them and not others I do not know.

A Physician: Aren't they most frequently associated with septic fevers?

Dr. Cabot: Yes, but we cannot make that

rule, because I have seen them in typhoid fever, where the variations are quite slight.

A PHYSICIAN: Do you get them without fever?

DR. CABOT: I should say it is rare to get them without any fever. But we do get them in alcoholism and in neurasthenia.

The pain in the upper right chest I do not think means anything in particular.

I ought to stop over the word "asthma." We use here and I think most people use "asthma" to mean paroxysmal dyspnea due to some external irritation or internal irritation, but not due merely to stasis. What is called "cardiac asthma" I think should be called cardiac dyspnea of a paroxysmal type.

His injections may have been adrenalin, but I think they were probably morphia. Adrenalin will ordinarily stop true asthma but not cardiac dyspnea. So I judge they gave morphia. Why they chose to give it in the abdomen I do not know.

In old men's or old women's winter cough it it always worth while to see if we cannot stop it with digitalis. A great many cases called chronic bronchitis on the evidence of a winter cough which disappears in summer are really passive congestion due to heart disease, and are really to be helped by digitalis rather than by cough medicines of any kind.

NOTES ON THE PHYSICAL EXAMINATION

"Well developed and nourished" is a phrase we use in a rather routine way. It ought to mean something. "Development" means that the person is as tall as he ought to be, is not undersized. "Nutrition" of course refers to the development of the fat layer.

It seems to me that two-thirds of all the patients that we go over in these exercises have a "marked pyorrhea." If I were inclined to think that pyorrhea was the cause of any particular disease I am sure I should have been disabused by this necropsy experience.

What else should they tell us about the thrill?

A PHYSICIAN: The time.

DR. CABOT: Yes. They ought to have said "systolic" or "presystolic" or both.

These are esentially normal kidneys.

I should say the important thing in the blood examination is a moderate leucocytosis.

The vital capacity is a good deal diminished, as we should expect it to be in heart disease.

I do not believe the lungs will turn out to have any disease other than congestion.

A PHYSICIAN: What value has the renal function test?

DR. CABOT: When it is zero we usually think of bad nephritis. When it is above five I do not think it means anything. Fifty per cent. is very high for a man in this condition.

They are trying hard to find mitral disease, and not finding it. A systolic thrill always makes us think of acute endocarditis with vegetations on the valve. They naturally therefore took cultures, which showed nothing, just contamination.

Testing the oxygen content of the blood is experimental work, not done for clinical purposes.

I did not see at first why they were feeling for the spleen, but they were thinking about emboli from a possibly acute endocarditis. The spleen would then be enlarged and there would be petechiae, but they did not get either sign.

The consultant assumes that it is infection.

This fibrillation is better than flutter. There is some risk in the use of quinidine because the heart has been fibrillating so long.

They looked for some cause of leucocytosis outside the heart but did not find it.

DIFFERENTIAL DIAGNOSIS

Of the types of heart disease this certainly does not look like hypertensive. He is rather young for it. We have no definite record of hypertension, and he must have had something else on account of his fever. We have no evidence of syphilis, or of the type of murmur that syphilitic aortitis gives if it produces any. We have an X-ray which rules out aneurysm. I think we can rule out syphilis. Rheumatic heart disease he may, so far as I know, have had. But I think that his ability to run as fast as he did run only twenty-one months before admission makes it rather improbable that he had a chronic deforming endocarditis. Many things suggest the fourth type of heart disease, the septic type, especially his temperature, his leucocytosis, the apex thrill, which is not a common thing in any other type.

The rest of the lesions ought to be passive congestion. So I should say this was either a rheumatic endocarditis or a rheumatic endocarditis with a septic on top. Against septic endocarditis is the fact that he has had no embolism. They have had only one culture, which is not much, and caught no bacteria in his circulation. I do not think the last ought to be considered much because they tried only once. He may have had embolisms that we did not discover.

A PHYSICIAN: What was the attack on February 14, shortly before he died?

DR. CABOT: I do not think we need to suppose any particular change. Any chronic heart case is likely to go through that stage when he gets near death.

A PHYSICIAN: Does this case suggest allergic asthma? Early in the history it says he had had three attacks twenty years before admission.

DR. CABOT: Yes, I had not noticed that. I think it is a good point. If he had asthma twenty years before admission the chances are it was not of the cardiac but of the ordinary type. Of course that does not show anything post mor-

tem, so it is not going to affect our particular efforts now.

There is another way in which this fever can be accounted for, and that should be mentioned. Intracardiac thrombi or extracardiac thrombi I believe can cause fever. All I know is that in many cases that have shown fever that is all that post-mortem examination has shown to explain the fever, and I do not see why small bits of thrombi breaking off cannot cause protein fever. We know that proteins can cause fever, and I do not know why they cannot be the cause here.

The diagnosis ought to rest between (a) rheumatic endocarditis with protein fever, (b) a rheumatic endocarditis with a septic process on top, or (c) a pure septic process. The only practical difference there is that if he had the last or the second we should feel that he could not get better. If he had the first he might get better.

A PHYSICIAN: The evacuation of a large amount of sputum at one time, with pain and night sweats, is suggestive of the evacuation of a cavity, although with this edema that he has it might be cardiac. Still it seems to me like a lung condition, although the X-ray does not show it.

DR. CABOT: And then if he did evacuate a cavity once he would go on.

A PHYSICIAN: Why did they examine the sputum so often? Was it simply because of his history?

DR. CABOT: I rather assume he did not spit while here. That was before he came. There was only one examination here. I think probably from that X-ray and from their own examination they thought there was not enough reason to think of tuberculosis.

A PHYSICIAN: Could this be some acute bronchopneumonia without any physical signs?

DR. CABOT: Yes, perfectly well.

A PHYSICIAN: It says he was toxic from digitalis. How would they find that out?

DR. CABOT: I take it his stomach was upset. That is the usual evidence.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Mitral stenosis.
Auricular fibrillation.
Auricular flutter.
Hypertensive heart disease.

DR. RICHARD C. CABOT'S DIAGNOSIS

Chronic endocarditis, rheumatic.
Acute endocarditis, septic?
Chronic passive congestion.

ANATOMIC DIAGNOSES

1. *Primary fatal lesions.*

Rheumatic heart disease—aortic stenosis.

2. *Secondary or terminal lesions.*

Hypertrophy of the heart.
Chronic passive congestion of the lungs, liver, spleen and kidneys.
Hemachromatosis.

DR. MALLORY: This is another case in which the clinical evidence was rather puzzling. So far as I know nobody made a correct diagnosis. They speak everywhere of mitral systolic and diastolic murmurs, but anatomically the mitral valve was entirely negative. On the other hand he did have a very marked old rheumatic aortic stenosis, which is the only lesion of the case. The heart was of course dilated and hypertrophied. The coronary arteries were negative. The heart muscle on microscopic examination showed a moderate amount of fibrosis, and all the other organs showed chronic passive congestion. As an accidental finding quantities of hemosiderin and small amounts of hemofuscin allowed us to make the unusual diagnosis of hemochromatosis.

DR. CABOT: How about intracardiac thrombi?

DR. MALLORY: There were none, and we were not able to give any cause for his fever and leucocytosis.

A PHYSICIAN: Why do you think he had the temperature?

DR. MALLORY: I don't know. Very commonly in these cases we get a terminal septicemia; but blood culture post mortem was negative. It is possible we failed to grow the organism. I think that is most likely.

CASE 13362

DYSPHAGIA, EMACIATION AND HEMOPTYSIS

MEDICAL DEPARTMENT

A colored Pullman porter fifty-six years old entered June 14 complaining of vomiting. He was extremely dull. The history is probably not accurate.

Two years before admission, when he was perfectly well except for an inguinal hernia, he began to vomit almost daily, usually half an hour to an hour after supper. There was no preceding nausea or heartburn and no retching or gagging. The vomitus consisted of a portion of the meal just eaten. There was no blood or coffee ground material. Several times each month after not vomiting his supper he would vomit the following morning and recognize food eaten the night before. He insisted however that he had vomited nearly every night for the past two years. Three months before admission he had an acute infection of some sort with a shaking chill, high fever, cough, sweats, and on one occasion some blood streaked sputum, coughed up, not vomited. He was in bed four days. After his convalescence he was "very nervous" and could keep nothing at all on his

stomach for three days. Castor oil gave great relief. Two months before admission he began to feel as though his food stuck midway down his gullet. He would be free from this feeling for a week at a time. When it was present solids and liquids gave equal trouble. The sensation lasted only a few minutes and never persisted until it had to be relieved by vomiting. Eight days before admission he felt so weak and "nervous" that he stopped work. From six to two days before admission he vomited practically everything he ate, usually half an hour to an hour after eating. The day before admission he vomited three or four times and the day of admission once. From June 6 to 12 his bowels did not move. The day before admission he took some castor oil with a resultant stool which was coal black and sticky as tar. He thought he had lost thirty-five pounds in the past two years and ten pounds in the past two weeks. He had grown weaker.

His family history is not significant. He gave a past history of "rheumatism" eighteen years before admission "all over his body, even in his eyes." No joints were involved. He had gonorrhea four times before he was thirty-six; no sequelae. Fifteen years before admission he fell twenty-five feet and was laid up for two weeks. For five years he had had a left inguinal hernia with coincident constipation, so that ever since that time his bowels moved only about twice a week and then only with castor oil. For three years he had had mild dyspnea on climbing two flights of stairs, had urinated two or three times at night, and had had occasional difficulty in starting the urinary stream and occasionally a forked stream. Until six years before admission he had been intoxicated twice a week. Until four weeks ago, when he took a high-ball, he had taken no alcohol for years. He had taken many kinds of patent medicine.

Clinical examination showed an undernourished Negro with slight bluish-grey pigmentation of the mucous membrane of the mouth. The lung signs were as shown in Figure I. The localization of the apex impulse of the heart is not recorded. There was no enlargement to percussion. A systolic murmur was heard. The radial arteries were thickened, the brachials tortuous. The abdomen was spastic. No organs or masses were felt. There was a right femoral and a left direct inguinal hernia. The pupils, knee-jerks and ankle jerks were normal. No clonus, Babinski or Kernig. Romberg was not done. The fundi were sclerotic.

The urine was normal in amount, specific gravity 1.020 to 1.030, cloudy at one of six examinations, no albumin or sugar, 3 to 75 leucocytes per high power field at all examinations, an occasional granular cast at one. Blood examination showed 5,400 to 11,500 leucocytes, 72 to 63 per cent. polynuclears, hemoglobin 60 to 80 per cent., reds 4,200,000 to 4,600,000, moderate achromia at one examination, reds normal at the other, platelets normal at both. Two Wassermanns were strongly positive. The sputum was purulent, positive for blood, negative for tubercle bacilli. The stools were negative at five examinations. Gastric analysis: The duodenal tube passed only to the 14 inch mark instead of the usual 20-25 inches, and persistently stopped there on several attempts. Fasting contents of stomach: 15 cubic centimeters of turbid water with a few food particles; no free hydrochloric acid, combined acid 2, guaiac negative. Test meal: 45 cubic centimeters of similar material with more whitish food sediment; no free hydrochloric acid, combined acid 3, guaiac negative.

X-ray June 18 showed partial obstruction to the liquid barium mixture in the middle third of the esophagus. At this point the esophagus appeared to be displaced slightly to the right and terminated in an irregular fashion. Above this area the esophagus was dilated and showed active peristalsis. Fluoroscopic observation and a film of the chest showed a rather diffuse increased density of the entire right midchest, most marked in the hilus. The lung markings radiating from the hilus on this side were increased in prominence and there was apparently some infiltration in the lung. The heart and mediastinal contents were displaced somewhat to the right. The right diaphragm was irregular. There were well defined adhesions between the diaphragm and the pleura. July 1 the appearance of the chest had not changed markedly since the last observation.

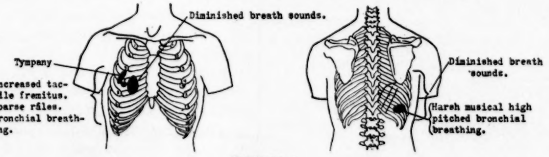
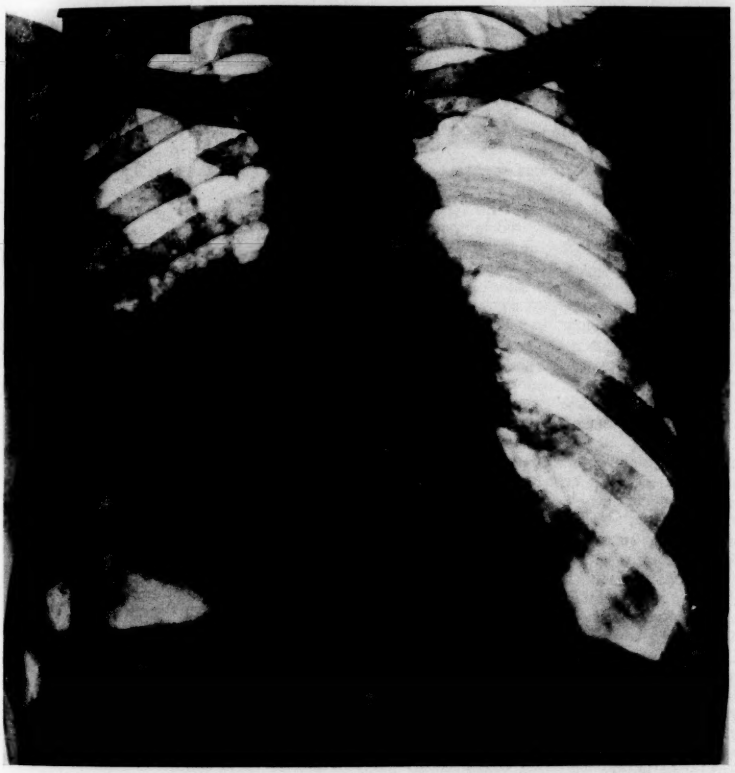


FIGURE 1

Orders. June 14. First week gastric régime. June 17. Magnesium sulphate one ounce. June 18. Soapsuds enema in the morning. Twenty to thirty minims of potassium iodide three times a day. June 23. Russian oil and cascara every morning and evening if necessary for catharsis. Second week gastric régime. June 24. Soapsuds enema. Magnesium sulphate one ounce.

June 25. Camphorated tincture of opium one dram; repeat in four hours if three bowel movements have occurred in the meantime, otherwise repeat in six hours. June 27. Paregoric one dram; repeat in two hours and again in two hours if dejections exceed four. June 30. Third week gastric régime. July 2. Heyden's solution ten minims every other day intramuscularly for four doses. Morphia sulphate one-quarter grain subcutaneously; repeat after three hours if necessary. July 3-8. Morphia in one-sixth to one-quarter grain doses.

The temperature was 97° to 102.8°, with afternoon rise throughout. The pulse was 70 to 146, the respiration 16 to 37.



Shows a rather diffuse increased density of the entire right midchest, most marked in the hilus. The lung markings radiating from the hilus on this side are increased in prominence and there is apparently some infiltration in the lung. The heart and mediastinal contents are displaced somewhat to the right. The right diaphragm is irregular. There are well defined adhesions between the diaphragm and the pleura.

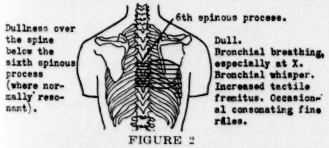


FIGURE 2

June 26 the patient was uncomfortable, possibly because of constipation. A lung consultant found the lung signs as shown in Figure 2 and found the heart one fingerbreadth inside the nipple line. He thought the abnormal spinal dullness interesting.

June 28 the patient was incontinent of feces and coughed a little blood. July 2 he expectorated about eight ounces of bright fresh blood. His pulse rose to 140. He brought up 40 to 80 cubic centimeters of blood daily for the next two days. July 5 examination of the chest with extreme care—the patient was not moved

—showed practically the whole of the right middle and lower lobes solid. That afternoon there was another hemoptysis. The following day there was no hemoptysis, but he was extremely weak and going downhill rapidly. July 9 he died.

Discussion

BY WILLIAM H. SMITH, M.D.

It is unfortunate that he was dull and the history probably not accurate. An accurate history is essential for a correct diagnosis; indeed oftentimes it is eighty per cent. of the diagnosis.

For two years the symptoms suggest regurgitation without pointing directly to gastric pathology, unless at times stasis, partial obstruction at a point near the cardia of too long duration for cancer; some condition along the cardia in the esophagus, possibly a diverticulum, possibly extraneous pressure. Three months before admission the symptoms suggest some acute infection, possibly pulmonary, too acute for tuberculosis. A month later the symptoms pointed to obstruction in the esophagus or cardia. The obstipation relieved by castor oil, associated with a tarry stool, makes strong the suggestion of carcinoma, especially if his observation of loss of some forty-five pounds in weight is true. The fact that this weight loss took two years and two months to occur is unusual, to say the least, in cases of cancer.

At thirty-eight "rheumatism" as here described certainly was not rheumatic fever. The possibility of some toxic periarthritic condition due to his chronic gonorrhea is tenable. A syphilitic manifestation should be thought of. There appears to have been no permanent legacy of his fall.

His dyspnea, mild on climbing stairs for three years, leads us to suspect some lack of integrity of his heart muscles or some pathological lesion associated with his arch or coronaries. No statement of angina pectoris or its absence appears. Although alcohol may have affected his myocardium, his abstinence for some years before his dyspnea would limit its bearing on the case.

His lung signs are malplaced for usual tuberculosis. They seem too localized for metastatic disease. They may be a legacy of his acute pulmonary infection, possibly an abscess, a bronchiectatic cavity, possibly compression from some tumor pressing on the bronchus, thus producing difficulty in swallowing.

With dysphagia and pathology in the lung in a patient with frequent Neisserian infection the possibility of syphilis of the arch with aneurysm should be considered. His history of dyspnea for three years would attract our attention to the heart; and certainly at fifty-six myocardial weakness, arteriosclerosis and syphilis should be excluded.

It is diffcult to interpret a systolic murmur when its location is not stated. It is difficult at times when the position of the murmur is placed. Arch sclerosis and syphilis of the arch may be impossible to differentiate in early stages, especially when there is no increase of the arch width. When this aortitis is placed near the aortic valve the diastolic murmur due to the aortic regurgitation strengthens the possibility of syphilis. Placing the patient on the face will at times bring out or accentuate the murmur. When one is attempting to exclude aortitis this same face position with the breath held will oftentimes enable one to discover a systolic murmur of wide distribution at the base. Aortitis must be eliminated or proved in cases with symptoms of dysphagia and pulmonary pathology, especially where the duration of the symptoms, twenty-six months, makes malignant disease less likely. The peripheral arterial sclerosis would be expected at this age from the acknowledged manner of living.

Some arteriosclerotic changes in the kidney would account for the urinary findings. The moderate achromia and other blood findings add nothing of value. The two strongly positive Wassermann reactions would make syphilis stand out prominently as against malignancy. The sputum examination does not help except to make tuberculosis of the lung unlikely, malignancy of the lung less in the foreground. Bronchiectasis or abscess is possible, the first from bronchial compression of a dilated arch, the second a legacy of his acute pulmonary infection. His pulmonary symptoms are in their acuteness unlike those seen in malignancy.

The point of obstruction, fourteen inches, met by the duodenal tube is of interest. But little help is obtained from the gastric analysis. Low or absent free hydrochloric acid may be present independent of cancer, or the patient may even appear to be in perfect health. The position of the obstruction of the esophagus, in its middle third, by X-ray examination, should make us think in terms of anatomy of the possibility of aneurysm. If mediastinal terms are to be considered, the chest condition undoubtedly masked a clear X-ray; from the description the differentiation would be difficult between a neoplasm from the hilus infiltrating the lungs or a chronic inflammatory condition. The degree of mediastinal displacement may have assisted in forming an opinion of malignancy or pneumonitis with pleuritis.

The progress of the case, with the extensive changes in the right chest, the dullness over the spine below the sixth spinous process, could be explained by rapidly growing malignancy of the lung, mediastinal tumor, infection or pneumonia, or hemorrhage in the lung.

I do not see how a differential diagnosis can be made accurately between neoplasm of the mediastinum with pressure on the esophagus and right lung invasion or aneurysm of the

aorta with compression sequelae. I am inclined to favor the latter because of the time incidence, twenty-six months, the positive Wassermanns and the well known danger of overlooking high aortitis with aneurysm when the symptoms are distant in the esophagus and the right lung. His tarry stools noted on one occasion may have been due to swallowing blood. I see no evidence of intestinal carcinoma.

INTERPRETATIONS OF X-RAYS

The changes in the esophagus are those of an obstructing lesion in the middle third. The picture is rather atypical for primary esophageal malignancy. It might well be produced by an extrinsic condition with secondary involvement of the esophagus.

The changes in the lung show a definite pathological process in the right lung root with apparent infiltration of the lung. Such a picture might be produced by primary malignancy, or by an unusual type of inflammatory process, possibly syphilis.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Carcinoma of the esophagus.
Terminal bronchopneumonia.

DR. WILLIAM H. SMITH'S DIAGNOSIS

Aneurysm of the aorta with compression sequelae, or
Less probably, malignant disease of the mediastinum with pressure on the esophagus and the right lung.

ANATOMIC DIAGNOSES

1. *Primary fatal lesions*

Luetic aortitis.
Aneurysm of the aorta with compression of the esophagus, trachea and bronchi and rupture into the right pleural cavity.

2. *Secondary or terminal lesions*

Extensive hemothorax, right.
Disintegration and hemorrhage and purulent infiltration of the right lung.
Decubitus.

3. *Historical landmarks*

Foci of obsolete tuberculosis of the left lung.
Chronic pleuritis.

DR. OSCAR RICHARDSON: He was a tall man, six feet two, poorly nourished.

The head was not examined.

Trunk. The abdomen was hollow, the muscles thin and pale. The peritoneal cavity and appendix were negative. The mucosa of the esophagus was negative; but the tube was pressed upon and considerably flattened at a point about at the level of the bifurcation of the trachea by an aneurysm to be described. The gastro-intestinal tract and the retroperitoneal glands were negative. The anterior margin of the right lobe of the liver was 8 centimeters—about a handsbreadth—below the costal border in the right mammary line—considerably below. The liver, gall-bladder, pancreas and spleen were negative, as were the adrenals, kidneys, prostate, seminal vesicles and testes.

The diaphragm on each side was at the sixth rib. This is low, especially for the right side. The right pleural cavity contained a very great amount of blood and blood clot. The left pleural cavity was obliterated by old adhesions; no blood. The parietal pleura on the right was markedly thickened in the region of the aneurysm, and there were some old adhesions about the lung. The left lung was bound down by old adhesions. The trachea and bronchi were pressed upon to some extent by the aneurysm and contained some blood and mucopurulent material. The bronchial glands were slightly enlarged and pigmented—negative.

The tissue of the right lung in the region of the lower half showed much infiltration with blood and purulent material and was disintegrated where it was in relation with the aneurysm. In the region of the lower part of the lower lobe and in the pleura adhesions along the paravertebral tissue there was a small collection of fibrinopurulent material. The left lung showed a few scattered masses of old tuberculosis, but was otherwise negative.

The outer surface of the pericardium was bound down by adhesions to the left parietal pleura just inside the left nipple line. The pericardium inside was negative. The heart weighed 330 grams. The mass of blood and blood clot in the right pleural cavity and the aneurysm pushed the heart over to the left so that the right hand border of the right auricle was in line with the left border of the sternum. The apex was at the sixth rib in the left nipple line. The myocardium was of good consistence and otherwise negative. The left cavity was negative, the right slightly dilated. The auricular appendages and the valves were frankly negative, the coronaries free, negative.

The wall of the ascending thoracic portion of the aorta beginning a short distance above the aortic valve was thickened in places, and there was much diffuse luetic-like fibrosis. This condition passed over into the wall of a huge aneurysm which rested in the region of the arch and extended for some distance down along the descending thoracic portion. It contained two very large masses of concentric layered thrombotic material, one of which was rather loosely attached, bee-hive in shape, and rested within the walls of the aneurysmal sac, from which it had extended to the right, pushing deep into the lung tissue, eroding, disintegrating it and rupturing into the right pleural cavity. The aneurysm extended downward nearly to the

upper border of the abdominal portion of the aorta. This portion of the aorta and the great branches showed only a slight amount of fibrous sclerosis. The microscopical picture of sections from several places in the region of the wall of the aneurysm showed the typical lesions of luetic aortitis.

Again we note that with typical luetic aortitis situated above the heart valves and not involving them there is lack of heart hypertrophy and dilatation.

BURBANK HOSPITAL, FITCHBURG, MASS.

By early October the new east wing at Burbank Hospital will be ready for occupancy and the new power plant and laundry in operation. The completion of this building program, plans for which were first considered in 1921, together with the necessary changes in the main group of connecting hospital buildings including vacuum heating system, automatic sprinklers and a new two-story kitchen-dining unit with modern equipment, represent an expenditure of $325,000.

The east wing, 225 feet long, provides with its two floors 37 private rooms attractively furnished, a 12-bed children's ward arranged on the cubicle plan, a three-bed ward, a two-bed ward with utility room connecting, for tonsil cases, two isolation rooms, diet kitchens, bath rooms and utility rooms. Generous window space throughout and delicate wall tints make it cheerfully light. Handsomely grained birch is the inside finish. On each end of the building are two spacious solariums and one of these on the north has airing platforms as well. Many of the rooms with easterly exposure get the full benefit of the wonderful and unusual outlook which Burbank's hilltop affords. The new 125-foot stack of the power plant locates the hospital from miles around. Across the broad valley one sees the famous Marshall apple farm.

The present main group of buildings consist of male ward, administration building, female ward and surgery. Their capacity under the new plan will be as follows: Male ward, 20 beds with five isolation rooms; administration building second and third floor, two and three bed rooms devoted to compensation and semi-private cases, 30 patients; female ward, 20 beds with five isolation rooms. The surgery consists of ample operating rooms together with well arranged laboratory and X-ray departments.

Total capacity including maternity hospital later referred to, will be 150 beds or a net increase of 60. This does not include the tuberculosis annex. For several years an overcrowded condition has existed; many people unable to get satisfactory accommodations have remained at home or gone elsewhere. Rooms intended for isolation purposes have been put to private use and too often there have been beds in the corridors. It is the sincere hope of the Building Committee that the increase will prove more than adequate. The impression persists, however, that the new space will rapidly be in demand.

The recent most generous gift to Burbank and the City of Fitchburg of the Lucy Helen Memorial Maternity Hospital by Mrs. Charles T. Crocker will permit greater service to the community than the plans in the new additions had provided, and already advantage has been taken of this service to its capacity. The property, conservatively valued at $100,000, is located in the central part of the city by the Upper Common and is conveniently accessible for physicians and visitors. The building, with spacious grounds, is in part newly constructed and finely equipped. Furnished almost exclusively, aside from beds, with well chosen early American pieces, it is both charming and unique. Its intended capacity of 20 mothers is adapted to increase. Besides a suite occupied by the managing supervisor, there are quarters for the necessary number of pupil nurses. The whole layout, and the administration of a smaller hospital as a separate unit lends itself to successful and efficient operation, pleasing to patients and most valuable and beneficial from the standpoint of training school service. Opened on February first, there were up to July first, 175 births.

The present nurses' home built in 1911 is overcrowded. The training school today numbers 42 with a graduate supervising staff of 15; of these 9 pupils and two graduates are at the Lucy Helen Maternity. By September the school must number at least 60 to properly serve the larger needs of the hospital. An incoming class of 20 probationers seems assured. The problem of the moment is additional quarters for pupil nurses. A plan under consideration is the closing of the tuberculosis annex built in 1914, the turning over to State care of the twenty odd patients and the taking of that building for a pupil nurses' home. It is a 28-bed unit and lends itself well for training school facilities. This procedure, however, is not altogether favored and some other method of accommodation may have to be worked out. At any rate, a larger training school maintaining the present high standard approved by both the Massachusetts and New York State Boards will be well and carefully provided for.

The hospital staff, comprised of 23 resident and 13 non-resident physicians and surgeons, gives a broad and efficient service. There is every reason to believe that with the more adequate facilities now realized and the close coöperation and harmony between administration and medical staffs, together with a training school with a spirit of service, Burbank Hospital moves forward with the purpose of providing the best possible care for those who may need its ministrations.

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Established in 1828

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ONCE MORE—DIPHTHERIA

A prophet would have to be a grim soul to find any satisfaction in the fulfillment of a prediction that added to the distress and sorrow of his fellow men. It is with no desire to gloat that we call our readers' attention to an editorial, "A Medical Duty," which appeared in this Journal in the issue of September 23, a year ago.

In discussing the lessened prevalence of diphtheria we said, "Unless the disease is changing its character—and that has never been its history—it will come back again and in virulent type." Apparently the respite is over—diphtheria is once more on the increase and it is returning in a more severe form. The prediction, unhappily, is being fulfilled.

The United States Public Health Service reports that for the first six months of this year, with the exception of the West, North Central and the Pacific States, there has been considerably more diphtheria in this country than for the same period in 1926. In our own State after having diphtheria fall in the summer of 1926 to one of the lowest levels in recent years, the number of cases began to mount in November, and show nearly a fifty per cent. increase for every month since. Reports from France, Germany and from various parts of our own country tell of the greater mortality from the disease.

While it is true that the present levels of diphtheria are well below those of years previous to 1926, and while with the immunization work already done and being done, the figures may not again mount to the higher peaks of the past, yet the disease so far seems to be running true to its historic form. Again, unless it changes its character, and all present signs fail, the increase in cases and deaths should continue until one of its periodic recessions again takes place, or until the medical profession, in more earnest fashion, sets about the task of its prevention through immunization.

If considered casually or with bias, it would seem that these figures furnish a hollow echo to the public health cry that active immunization with toxin-antitoxin is bringing down the rates. We have more than once objected editorially to some of the boastful claims made by not a few public health enthusiasts and we have little sympathy with those who promise to eradicate diphtheria by 1930. We are convinced, however, that diphtheria toxin-antitoxin mixture is as efficacious as it always has been. It protects the individual against the disease, and where sufficient individuals are thus protected diphtheria cannot prevail. Thus the population of an institution, of a community, or of a State or country can be permanently shielded against this disease. We have had ample proof that this conviction is sound and the diphtheria returns for the year only strengthen it. The increasing diphtheria is attacking the non-immune children and it is the communities with the smallest number of protected children that are contributing more than their share of cases.

While it is certain that as yet an insufficient portion of the State or country's child population has been immunized to account wholly for the low rates of the past two years, it is now apparent that many communities where sufficiently large numbers of children have been protected are not sharing in the greater prevalence and are helping to limit the general increase in their part of the country. Their present happy security will, however, continue only so long as they continue the use of toxin-antitoxin.

The 1927 figures, therefore, refute the extravagant claims of some unthinking enthusiasts, but affirm the claims of the conservative and thoughtful sanitarians that a parent or a community can determine whether or not diphtheria is to be avoided.

It seems, however, that this lesson is never fully learned. Here is an easily avoidable disease with the means for its prevention freely available, and yet diphtheria goes on turning its cycles and taking the lives of susceptible, unprotected children.

The situation is one that, because of its constant urgency, should arrest the attention of all medical men. The responsibility after all is

theirs. They have it in their power to protect the children entrusted to their care, and they fall short of their duty unless they make every effort to persuade parents to permit their children to be immunized. With the immunization of all susceptible children and the regular practice of giving toxin-antitoxin to every baby on its first birthday, the day is bound to come when diphtheria will find its way blocked to our homes and our communities. In this connection we take occasion to repeat the warning published in the *Transcript* of the 30th ult.:

MAYOR NICHOLS ASKS PARENTS TO TAKE THEIR CHILDREN TO THEIR FAMILY PHYSICIAN FOR IMMUNIZATION OR TO HEALTH STATIONS

There has been an increase in diphtheria cases since the beginning of the year, and Mayor Nichols, after consultation with the Health Department, issued a statement today, in which he advises parents to see that their children are immunized, either by their family physician or at the health stations. The statement is as follows:

"According to the reports which I have received from the Health Department there is an increase in diphtheria cases during the past seven months of 1927 as compared with the same period of the previous year. Statistics show that there is a greater proportionate increase all over the United States.

"The Health Department of the city of Boston has well organized plans for the prevention of this disease and it is disappointed to observe this increase. The injection of a harmless fluid resulting in almost every instance in the establishment of immunity makes it possible for all parents to protect their children against diphtheria.

"I earnestly urge all parents to take their children to their family physician for immunization or to one of the thirty weekly clinics which are conducted by the Health Department of the city of Boston.

"CANCER CURES"

THERE is no group of diseases in which the medical profession, taken as a whole, appears to be more helpless than in the case of cancer. There are some cases cured by surgery; there are some cases cured by radium; there are some cases cured by X-ray; there are undoubtedly cases whose death has been hastened or whose end has been made far more painful by the unskilled use of these various methods. This is particularly true of radium and X-ray. There are many cases of malignant disease obviously hopeless from the start. There are others which may be relieved or even cured.

The treatment of cancer by chemical methods has been left largely to quacks. There are cases of superficial cancers of low malignancy which have undoubtedly been healed by the application of various escharotics and caustics. There are vast numbers of cases whose misery has been increased by these substances. Is it possible that in the case of a disease, such as cancer, where the orthodox measures are admittedly none too satisfactory, that we are a little too complacent? Is it not possible that some good thing may be learned from the Ishmaelites?

When we consider that empiricism has preceded many of our scientific discoveries we may have been shutting our eyes to possible avenues of attack on the tremendous problem facing us in malignant disease. It is a pity that there is not some authorized and recognized agency which can, without committing itself or without undue publicity, try out some of the more promising of the unorthodox remedies. Certainly even some of the crude arsenical preparations could do no more, if as much, harm as does radium in unskilled hands.

The medical profession knows too little of cancer as a terminal disease, sees too little of the end results of treatment, particularly by radiation. If, among the unrecognized "cures" there are any even half as effective as radiation, providing they were only half as dangerous when improperly used, their adoption by the medical profession might be an advance. Unfortunately, most of the existing agencies for the investigation of new remedies have, perforce, surrounded themselves with an almost impenetrable barrier of ethical considerations. If for nothing else but to relieve the public, preyed upon by the purveyors of fake cancer "cures," these "cures" should be investigated by some disinterested group and the results given widespread publicity.

TYPHOID VERSUS PASTEURIZATION

TYPHOID fever, a water-borne disease before the days of modern water filtration, is now a milk-borne disease, according to Dr. S. J. Crumbine, Field Secretary of the Conference of State and Provincial Health Authorities of America. Formerly unsafe water supplies and inadequate sewage disposal were the chief causes of our annual epidemics of typhoid fever, and typhoid fever was then largely a disease of cities; cities are now safer to live in than is the country, as far as typhoid fever is concerned, for the main sources of the disease in cities—water and milk—have been rendered safe.

It is in the rural districts where milk is not sterilized and where its production is not inspected that typhoid still prevails in any degree. The disgraceful experience of Montreal during the year of grace 1927 is a notable exception and shows what may happen in a large modern city with a mediaeval public health service.

Water-borne typhoid is now rare. Contact infection and contaminated foods, in the contamination of which the house fly plays an im-

portant role, are the chief sources of the sporadic and endemic forms of the disease. Milk has become the source of most of our modern epidemics, the record for 1926 so far tabulated showing 37 States reporting 47 epidemics that were milk-borne.

History is repeatedly forcing upon our consciousness the fact that the only absolutely safe milk—not only as regards typhoid, but also as regards the other infections that may be milk-borne—is an effectively pasteurized clean milk. The demand for such a milk is widened by the fact that some dairymen are producing a certified milk and then pasteurizing it, which would seem like painting the lily but is in reality an effort to provide an ideal milk. If this State is to maintain its high rank in sanitation and in public health efficiency we should all coöperate with our Commissioner of Public Health in attaining the goal of universal pasteurization. When universal, effective pasteurization of milk goes into effect, milk will pass out of the picture as a source of epidemic disease.

GAS MASKS

THE originator of the saying, "Let George do it," must have known a fireman named George. Whether it's a kitten up a tree, an explosion of a still, a fallen live wire, or a burning house, our first instinct is to call the fire department, and the fire department does its job when it is called.

In metropolitan Boston where the gas company has its emergency crews and equipment of all sorts is available for practically every conceivable emergency, there should be little, if any, difficulty in meeting any situation which may arise. But even here, the tragedy due to ammonia fumes at the Arena is an example of an emergency that could be handled only at a sacrifice. In our rapidly growing suburban communities hazards multiply faster than the means to meet them. Thus, we have in mind a town where gas is being installed for the first time, where undoubtedly there will sooner or later be deaths incidental upon its use.

The question came up as to whether the ladder company of the fire department of this town should be provided with gas masks in order to equip them for rescue work. They had gas masks suitable for smoke, but apparently not suitable for gas or ammonia fumes. There is, apparently, no one authoritative statement available as to what type of mask is advisable for any given type of hazard. A mask that will not protect against a certain kind of gas or fumes is worse than none at all, because of the false confidence it gives to its wearer. Would it not be well if a simple statement could be drawn up as to types of protection available against the different types of hazards presented by smoke, gas and fumes; and made readily available for the use of those whose work calls them into all kinds of unexpected dangers?

Again, the question of use of the Schaefer method of resuscitation as against the pulmotor or other mechanical devices was argued. Unfortunately, it seems necessary to remind physicians called for cases where resuscitation is necessary, that mechanical devices for artificial respiration are far from satisfactory, and that the Schaeffer prone pressure method of artificial respiration should be followed for at least three hours before giving up hope. We have more than once seen well-meaning physicians attempt to substitute mechanical devices for the intelligent methods of the victim's fellow workers. It would not be far from the truth to say that the average electric lineman knows more about resuscitation than some physicians.

A QUESTION OF DISCIPLINE

THE following question sent by letter to the Chairman of the Board of Registration in Medicine has been submitted to the JOURNAL: "Can a physician's license be revoked for immoral practices committed on a person other than a patient, if he is proven guilty? If so, does the Boston Medical Society deal with these cases or does it have to be taken to a Civil Court?"

It is our opinion that under our law the Board of Registration may revoke the registration of a physician who has been convicted of a felony, or is guilty of any crime committed in connection with the practice of medicine.

Before taking action the Board must conduct a hearing at which the accused may be present or be represented by counsel, if he so desires.

Any person cognizant of any irregularity on the part of a physician ought to present the facts to the Board of Registration in Medicine and the Board will determine whether the complaint warrants action. Even if no action can be taken all the facts should be in the possession of the Board for reference in case the alleged offender should come before the Board on some other charge. Accumulated evidence of objectionable conduct may be of value in enabling the Board to form opinions with respect to the behavior of a doctor under investigation. The Board is not confined to the rules governing court procedure as far as the consideration of evidence is concerned, and may with propriety take into account facts which may have a bearing on the general reputation of a doctor, but appeal may be taken concerning any action by the Board which tends to deprive a practitioner of the privileges conferred by registration. Appeals properly drawn and based on sound arguments will be considered by the courts which have the prower to overrule action by the Board.

So far as action by medical societies is concerned, such organizations have no authority beyond advising, censuring or expelling a member.

The writer of the letter should of course know that opinions even of lawyers do not always interpret laws correctly. The final decisions are made by the Supreme Court and questions may arise in connection with a given case which may need clarifying interpretations by this body.

If the questioner knows of any improper behavior of a doctor and he is willing to submit evidence tending to conviet a doctor and stand behind it, the matter can be adjudicated either by the Board of Registration in Medicine or by the Courts.

Often the difficulty lies in the indisposition of a person who knows of bad conduct to present the facts to the proper authorities.

Since the above was written a similar request has come to us with another question which is as follows:

If a surgeon deliberately fails to register the birth of an infant is this sufficient, if proven, to revoke his license?

The law provides that births shall be recorded by the attending physician if he is in charge.

If the attending physician wilfully neglects this duty and repeats the offence the Board of Registration could revoke or suspend his license but such action would in all probability be considered rather harsh unless the offence is repeated after censure.

Doctors are sometimes preoccupied and like other people forget to perform a proper duty but there is very little reason to feel that any accoucheur would deliberately defy the law. If he does he can be disciplined according to the interpretation of his behavior by the Board of Registration.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

GAMBLE, J. L. A.B., M.D. Harvard 1910, Associate Professor of Pediatrics at the Harvard Medical School. His subject is: "The Chemistry Findings in Rickets." Page 373. Address: 33 Edge Hill Rd., Brookline.

WYMAN, EDWIN T. M.D. Tufts College Medical School 1911, Instructor in Pediatrics Harvard Medical School, Assistant Physician to the Children's and Infants' Hospitals. His subject is: "The Prevention and Treatment of Rickets." Page 376. Address: 483 Beacon St., Boston.

MORSE, JOHN LOVETT A.B., A.M., M.D. Harvard Medical School 1891. Professor of Pediatrics Emeritus, Harvard Medical School, Consulting Physician at Children's and Infants' Hospitals, Member: New England Pediatric Society, American Pediatric Society, Association of American Physicians and Boston Obstetrical Society. His subject is: "A Plea for Sanity in the Use of the Modern Methods for the Prevention and Treatment of Rickets." Page 388. Address: 483 Beacon St., Boston.

MISCELLANY

DR. DRAPER TO STUDY GERMAN CONDITIONS

DR. W. F. DRAPER, Assistant Surgeon General in charge of the Domestic Quarantine Division, Bureau of the Public Health Service, will sail for Europe on September 6 to study public health methods in Germany for two months. Announcement to this effect was made by Dr. C. C. Pierce, Assistant Surgeon General, who explained that Dr. Draper's trip is part of the system of international interchange of public health officials inaugurated several years ago for the purpose of keeping health officials in each country acquainted with advances in their field made by the health officials of other nations.—*U. S. Daily.*

OBITUARIES

THE PASSING OF THE MANAGING EDITOR OF *AMERICAN MEDICINE*

ON Saturday, August 6, 1927, Dr. H. Edwin Lewis, who had been Managing Editor of this journal for nearly twenty years, passed away at his home, 86 South Highland Avenue, Ossining, N. Y., to which the town he had moved only a few months previously, following the destruction by fire of Rockledge, the family home at Searborough, N. Y., where he had resided for eighteen years. In Dr. Lewis, *American Medicine* loses not only an able and courageous editor whose independence of opinion, fairness to all, and outstanding capability as a medical writer gained him the esteem alike of his friends and of those who did not agree with him, but it loses also a chief who was a man in the truest and worthiest sense of the word, who, despite the unusual handicap of physical suffering under which he labored for many years, had always a cheery word of encouragement, an open smile, a hearty handshake for all with whom he came in contact. Born in Providence, R. I., February 22, 1875, H. Edwin Lewis received his early schooling in that city and attended Brown University until 1895 when his parents, Charles Edwin and Alla M. Lewis, moved to Burlington, Vt., where he continued his medical studies at the University of Vermont, graduating in 1897. Always of a literary turn of mind, Dr. Lewis founded the *Vermont Medical Monthly* shortly before his graduation and acted as its editor for a considerable time thereafter. While practicing general medicine in Vermont, Dr. Lewis specialized in nose and throat work and

tuberculosis. So clearly did he demonstrate his abilities in that direction that he was appointed secretary of the Vermont State Tuberculosis Committee, in which capacity he aided materially in framing the State Tuberculosis Act. In 1901 he was sent as a national delegate to the British Congress of Tuberculosis held in London. He embraced the opportunity afforded him to study and observe the methods in vogue in the leading hospitals of London and Paris. Returning to this country he continued to practice in Burlington and served as house physician to the Fanny Allen Hospital in Winooski, Vt., a suburb of Burlington, likewise enjoying extensive post-graduate study in Montreal under specialists in tuberculosis. In 1906 he moved to New York City, abandoning a growing practice for the sake of the wider field of usefulness he saw opening up to him as Editor of the *International Journal of Surgery*. Two years later the opportunity presented itself to take over the management of *American Medicine* which had gained a national reputation under the direction of Dr. George M. Gould. This reputation Dr. Lewis not only kept up but enhanced through the unusually happy combination he possessed of sound medical knowledge, literary skill and organizing ability. At the same time he continued to practice, serving for a while as surgeon to the Harlem Hospital and Attending Physician to the New York Throat and Lung Hospital (now the Midtown Hospital) and later acquiring considerable reputation as a diagnostician.

Dr. Lewis' personality, knowledge, and literary ability rightly won for him many honors. He was president of the American Medical Editors' Association, a Fellow of the American College of Physicians, a member of the New York State and County Medical Societies and a Fellow of the American Medical Association. He also occupied a high position in the Masonic Fraternity, having taken his degree through the York Rite to become a member of Cairo Temple A. A. O. N. M. S. in Burlington and Rutland, Vermont. In later years Dr. Lewis' life was largely a struggle against the handicaps of ill health and disabilities over which he had no control. That he succeeded in spite of obstacles which would have seemed unsurmountable to many is but another tribute to his indomitable courage and strong personality.—*Excerpt from the Obituary of Dr. H. E. Lewis—American Medicine.*

RESOLUTIONS ON THE DEATH OF DR. HERBERT E. BUFFUM

At a meeting of the Somerville Medical Society held August 26th, 1927, the following resolutions were passed:

In the death of Dr. Herbert E. Buffum the Society has sustained the great outstanding loss of one of its most able and eminent members. By his unfailing courtesy, his sympathetic and understanding humor, and his helpful and wise co-operation and leadership in all that pertained to the good of the profession, he had endeared himself to all.

We realize keenly the loss of this public spirited and high minded citizens to the community at large, but feel that it falls with especial force upon his patients and professional brothers.

We extend our most heartfelt sympathy to the bereaved family.

Be it further resolved that these resolutions be spread upon the records of the Society, and a copy sent to the family, and also a copy to the *Somerville Journal* and the Boston Medical and Surgical Journal.

(Signed) Frank E. Bateman,
Henry F. Curtis,
Eugene L. Maguire,
Committee.

CORRESPONDENCE

CRITICISM OF THE PUBLICATION OF AN ARTICLE ON THE TOOTH BRUSH

Health Department
City of Newton, Massachusetts
City Hall, West Newton

August 19, 1927.

Editor, Boston Medical and Surgical Journal:

In a recent number of the Journal there is an extract from the *Bulletin of New Haven, Conn., Health Department* giving the account of a preliminary study of the bacteriology of the tooth brush, which I read with great wonder and the more I read, the more my wonder grew.

Two questions presented themselves for solution:—Why the New Haven Health Department undertook the "study"? and why the Journal printed it?

I fail to see what value such a "study" can have for a Health Department. It cannot be for the protection of the public, for one cannot imagine any article, the use of which is more restricted to the individual, than a tooth brush, even though, in the "study" three were promiscuously used, which probably means they were family brushes, it being inconceivable that they were loaned to visitors.

This being so, the tooth brush as a transmitter of infection from person to person outside of the family may be ruled out. Among the members of those families who use "family brushes" the intimate relations existing offer many more probable methods of transmitting infection, so that again, the dangerous role of the tooth brush is negligible. Neither can a knowledge of the bacteria found in any given brush be of any value in protecting the owner from infection, as he is the original source of whatever organisms may be found in it.

With the sole exception of the identification of the varieties of bacteria residing in tooth brushes, which is interesting from an academic point of view, the "study" showed nothing that might not be predicated. Old brushes, brushes improperly cared for and brushes used by persons with dirty or decayed teeth all showed higher counts than new brushes, those more carefully cared for and those used by persons whose teeth were in good condition, all of which might be expected. Unused brushes, exposed to the air and presumably handled freely by would-be purchasers,

showed the presence of bacteria; again, a not unexpected result.

It seems to be that the whole "study" is an instance of misdirected energy on the part of a Health Department and one which is liable to bring discredit upon Health Department activities.

There are many useful lines of research which Health Departments with spare time and money can undertake, and, for this reason, I am surprised that a Department with the reputation of that of New Haven should have undertaken such a "study" as the one under consideration.

FRANCIS GEO. CURTIS, M.D.,
Chairman, Newton Board of Health.

EDITORIAL NOTE:—We would like to remind Dr. Curtis of the fact with which he is probably familiar that there is some criticism of present-day tooth brushes and the use thereof founded on bacteriological study.

Perhaps Dr. Curtis will agree that infections may follow self-inflicted abrasions.

We are not committed to the abolition of the tooth brush but we can see possible benefit accruing from knowledge of its vehicular function in its relation to bacteria.

It will do no harm if tooth brushes are recognized as carriers and receive attention according to standards of cleanliness.

A CORRECTION OF THE DATE OF OPENING THE ADAMS NERVINE

807 East Windsor Road, Glendale, Calif.,
August 25, 1927.

Editor, Boston Medical and Surgical Journal:

In your editorial "Fifty Years of the Adams Nervine" in the JOURNAL of August 18, you state "patients were not received in the hospital until 1888."

As I was connected with that institution before 1888 I would like to correct your statement. From July, 1886, to July, 1888, I was Assistant Physician under the late Dr. S. G. Webber, and if my memory serves me right there were admitted to that hospital about one hundred and fifty patients during my last year of service.

Perhaps some one may make this correction before my letter reaches you from this distance.

Very truly yours,
E. L. FISKE.

EDITORIAL NOTE:—Dr. Lane, superintendent of the Adams Nervine, reports that the first patient was received in 1880.

WORKMAN'S COMPENSATION STATISTICS

Everett, Mass., August 17, 1927.

Editor, Boston Medical and Surgical Journal:

During the last few weeks the writer has accumulated on his desk, for wet weather reading and study, dry statistics found in the latest printed annual reports of several State Departments. A list is found at the end of the chapter.

The first thing noted was that most of the data were two or three years old and that the statisical year of the different reports corresponds for the most part, neither with that of other departments or the calendar. As far as possible the findings apply to 1925, some of the data sought for the year is still in the hands of the printer. Under these conditions the findings can not have the present day accuracy of a railroad time table, which does not always guarantee that the train will be on time, though a late train may arrive if given time.

In many instances the data are approximate only and seen from the view point of a general medical practitioner rather than a statistical expert.

During 1925 nearly one million workers were protected by insurance; 59,000[1] or 6% were sufficiently injured to be officially recorded, 308 of the injured died.

Of those with temporary disability, 20,175, or 35%, were disabled seven days, or less; 22,906, or 40%, were disabled four weeks, or less. In 75% of the cases the disability did not exceed 28 days.

There were 33 stock companies with an expense ratio of 40.47%, and 12 mutual companies, with an expense ratio of 19.86%, that provided the insurance for the employees. During years '22, '23 and '24 the stock companies did 57% of the workman's acident compensation business, and the mutual companies 43%.

During these years four companies did over 60% of the entire business. For the three years these were the Liberty Mutual, Employer's Liability, Travelers, and American Mutual in the order named.

Among the whole number of employees there were about 200 classified groups. Among the fatalities of 308, 172 occurred in three groups with an aggregate of 23,314 workers. Buildings trades with 60 deaths among 8,550 workers; Transportation, 79 deaths, with 6,405 workers; and Trade, 33 deaths, 8,359 workers.

The most costly occupations for '22 to '24, Table Z[2], were Ice Harvesting with an average cost of $7.16 per $100 pay roll; Iron and Steel Construction, $7.06 per $100 pay roll; Junk Dealers, $7 per $100 pay roll —out of 22,890 protected workers.

The least expensive were Clerical Office Employees, $.01 2/3; Professors and Teachers, $.03 2/3; and Salesmen, $.04 2/3, each per $100 pay roll, with a total of 26,420 protected workers.

The premiums collected by the 45 insurance companies in 1924, Table Y[2], was $13,029,000 on an audited pay roll of $1,365,000,000. The average premium rate being a little under 1% or $.955 per $100 pay roll. The average loss for all companies in '24 was $.55 per $100 pay roll: $.59 for the stock companies and $.51 for the mutuals. The average premium for all companies, '22, '23 and '24, was $.8757 per $100 pay roll.

The average loss incurred for the three-year period was 63% of the audited premiums in stock companies and 61 2/3% in the mutuals. If the 40% of the premiums is a legitimate overhead charge for the stock companies where is the profit, and if the business is not profitable why pay 17 1/2% to get more business?

Table II[1] shows $7,327,000 paid for injured workmen in '25. From this $2,365,000 was paid for medical services; $919,000 paid for fatalities and $4,044,000 paid for lost time. While one injured employee out of 192 died, the cost of his injury was 30 times the average non-fatal case and the liability of the 308 dead was one-eighth of the whole cost of the 59,000 injured.

While the medical payments for '25 were 32.3%[1] of the whole payments, they were only 18% of the premfums collected. To the insurance companies medical and hospital service is as valuable financially as the work of agents acquiring new business. This has been referred to before in the JOURNAL but is still worth considering.

The fact that the law still recognizes the injured worker's right to choose his own physician is good evidence that the public looks upon the care of the worker as something more than a business proposition. The health and well being of the individual is a very personal matter and for serious injuries the worker rightly demands a personal physician who can be held personally responsible for what he does. Who is responsible legally in public clinics, in large hospitals connected with medical schools, or insurance clinics? From the workers' and doctors' view point the injured employee is something better than an unboned piece of meat to be whipped into working shape as soon as possible by machinery.

As the writer interprets the opinion of the Industrial Accident Board, expressed in its 6th An. Rep., p. 54, the assumption is made that any added expense to the insurer would be passed on to the employers, who in turn would pass it to the public, in the added cost of the manufactured product. This of course presupposes that no farther business economics are possible either by the insurers, or employers; and that safeguarding against accidents by employers and employees has reached perfection; and that no better coöperation between workers, employers, insurance companies and doctors would have lessened the death rate among workers, or made the period of disability shorter.

The voice of the public is scarcely heard unless stimulated by propaganda. Every public expense should be carefully investigated, whether a doctor's fee or the commission of an insurance agent. In the public service, is not the question whether the public gets what it pays for as important as the item itself? Added expense in some directions often saves greater expense in other ways.

The Compensation Law as amended this year, Sec. 30, says that "Medical services shall be adequate and reasonable and the charges reasonable." While insurance is an expert business that should be run for a legitimate profit, the medical cure, care and rehabilitation of the injured employee is a medical matter which medical men should have a directing hand in accomplishing. Who can decide this technical matter, what is adequate service and reasonable medical charges? Medical men generous with medical charity, or business men seeking a profit?

The law gives the Industrial Accident Board the right to decide what is "Adequate and Reasonable." Without some light from a Medical Advisory Committee with a technical knowledge of medical matters, how can members of another profession be sure to decide justly? Would the lawyers of Massachusetts think it fair for a board of doctors to tell them what was a reasonable charge for legal service given the public?

Table IX[1] deals with infection. There were in 1925, 5021 cases of infection or 8.5% of the tabulated injuries with 31 deaths, 10% of the whole number of deaths. There were 79% of the deaths by infection among 3995 cases of minor surface injuries yielding 24 deaths. The report of the investigation conducted by the Department of Labor reported that the blame lay with the employees who failed to use available protective measures. From a medical standpoint the investigation should have gone farther than the record shows. Certainly a very proper question to refer to a Medical Committee. We are informed there is such a committee that has not been called to function recently.

The Special Committee of last year, p. 21[2], recommended an increase in the compensation given the injured worker and estimated the increase would add "6.8% to the cost of the act." The Legislature in large measure carried out this recommendation. It may be of interest to see how much this added expense would affect the manufactured products of the State if applied without loading. Using the data for '25 given on p. 56[4], there were 593,000 wage-earners employed constantly in the combined Manufacturing Industries in the State.

The wages paid were $719,000,000 and the value of the manufactured products was $3,451,000,000. Using the premium of '24, a later premium not being available, the addition of this premium rate applied to the actual pay roll of '25 would add $6,867,000 to cost of production. This would add $.002, two mills, to each dollar of products and would yield an advance of $.20 on each $100 worth of production. The labor report mentioned gives $1,212 as the average yearly wage of manufacturing workers. His share if the distribution among all products was in equal proportion, would cost him $2.43 a year under the premiums of 1924: $2.43 being his contribution for protection under Workmen's Compensation. If the 6.8% be added under the 1927 law, his contribution would be raised $.16, totalling $2.59 for a year's insurance.

Again using the data in Table Y[3], the total premiums collected in 1924 by the 33 stock companies were $7,048,947; losses incurred, $4,260,755; leaving a balance to the companies' use of $2,788,192. The share of the companies being 65% of the benefits received by the employees. The public through the employers contributes $13,000,000 to benefit the employees and the Insurance Companies act as public servitors, *for a profit*. For every dollar paid the employee, the doctor, the hospital or dependents of fatalities, the stock companies take their toll of $.65.

Table II[3] shows the medical and hospital service for the year ending June 30, 1925, cost the State $2,364,580. Of this liability the stock companies' share of 57% would be $1,347,810. Comparing this sum with the $2,788,192, the share of the stock companies for their part in administering the act, we find the cost to the State more than double the cost of the medical and hospital service rendered under stock company supervision.

Table II[1] gives $919,000 paid in 1925 for fatalities with dependents. If the ratio of 57% liabilities and 65% toll still applied, the toll for the stock companies would be $340,450 on this group of cases alone. Does this seem adequate? Does it look reasonable? Public contribution of $1.65 to secure $1.00 of benefit? What is the answer? Three of the five members of last year's commission considered some other method desirable, two in a minority report advocating "Compulsory Insurance," one, a Monopolistic State Fund.

There are several matters more vital to the doctors of the State but this chapter is already too long. If anyone cares for another chapter let the writer know.

GEORGE E. WHITEHILL, M.D.

REFERENCES

1 An. Rep. Dept. of Industrial Accidents, 1925.
2 Sixth An. Rep. Dept. of Industrial Accidents, 1918.
3 An. Rep. Commissioner of Insurance, 1925.
4 An. Rep. Dept. of Labor and Industries, 1926.
5 Rep. Special Commission on Operation of Workmen's Compensation Law, '26.

CONNECTICUT DEPARTMENT OF HEALTH

MORBIDITY REPORT FOR THE WEEK ENDING AUGUST 22, 1927

Diphtheria	15	Encephalitis, epidemic	1
Last week	33	Influenza	1
Diphtheria bacilli carriers	5	Mumps	3
Typhoid fever	5	Pneumonia, lobar	12
Last week	3	Poliomyelitis	17
Scarlet fever	13	Tetanus	1
Last week	8	Tuberculosis, pulmonary	25
Measles	6	Tuberculosis, other forms	2
Last week	6	Chancroid	1
Whooping cough	28	Gonorrhea	34
Last week	33	Syphilis	64
Bronchopneumonia	1		
Chickenpox	10		

NOTICES

DR. HYMAN S. QUEEN HAS MOVED HIS OFFICE

Dr. Hyman S. Queen has moved his office and residence from 54 Russell Street to 246 Pleasant Street, New Bedford, Mass.

DR. STEPHEN RUSHMORE announces that he has withdrawn from administrative work and will devote himself to his practice.

NEWS ITEMS

RESIGNATION OF DR. CHARLES AARON DREW—Dr. Charles Aaron Drew, who has served the city of Worcester many years as superintendent of the City Hospital, has resigned and will engage in private practice. Dr. Drew was formerly a member of the Massachusetts Board of Registration of Nurses.

NOMINATIONS OF PHYSICIANS BY HIS EXCELLENCY GOVERNOR FULLER—Governor Fuller has nominated for consideration by the Council the following named persons: Dr. Frederick R. Barnes of Fall River for the position of medical examiner, succeeding Dr. W. H. Blanchette; Dr. Barnes is a graduate of Temple University School of Medicine of Philadelphia, Pa. Dr. Irving J. Walker of Boston for membership on the Board of Registration in Medicine, succeeding Dr. Birnie of Springfield; Dr. Walker graduated from the Medical School of Harvard University in 1907.

In all probability these nominations will be confirmed by the Council.

REPORTS AND NOTICES OF MEETINGS

THE ANNUAL MEETING OF THE NEW ENGLAND SURGICAL SOCIETY

THE Annual Meeting of the New England Surgical Society will be held September 30 and October 1, 1927, in Manchester, New Hampshire, with Headquarters at the Hotel Carpenter. A tentative program of the scientific papers has been prepared which is appended. The local committee, under the chairmanship of Doctor Wilkins, will provide interesting clinics and enjoyable social functions.

TENTATIVE SCIENTIFIC PROGRAM FOR ANNUAL MEETING NEW ENGLAND SURGICAL SOCIETY

September 30 and October 1, 1927

1. Conservatism in Renal Surgery, Dr. J. D. Barney.
2. The Desirability of a Second Removal of the Prostate in Certain Cases of Carcinoma, Dr. A. L. Chute.
3. The Treatment of Compound Fracture, Dr. P. P. Swett.
4. Heliotherapy and the Treatment of Surgical Tuberculosis, Dr. N. Allison.
5. Fractures of the Odontoid Process, Dr. R. B. Osgood and Dr. C. C. Lund (by invitation).
6. Acute Pancreatitis, Dr. R. P. Watkins.
7. Acute Pancreatitis, Dr. E. L. Hunt.
8. Repair on Hernia Work Under Local Anesthesia, Dr. D. C. Patterson.
9. End Results of Chronic Cholecystitis, Dr. E. L. Young, Jr.
10. The Unrelieved After Operation, Dr. A. G. Rice.
11. Sarcoma of the Uterus, Dr. R. H. Miller and Dr. H. Rogers (by invitation).
12. Radical Pelvic Surgery, Dr. H. T. Hutchins.
13. The Surgery of Pulmonary Tuberculosis, Dr. W. Whittemore.
14. Enterostomy in Peritonitis and in Obstruction, Dr. H. W. Clute.
15. Intussusception, D. W. E. Hartshorn.

THE MEETING OF THE SEVENTEENTH CLINICAL CONGRESS OF THE AMERICAN COLLEGE OF SURGEONS

THE American College of Surgeons will hold the seventeenth Clinical Congress in Detroit, October 3-7. Headquarters will be at the Book-Cadillac and Statler hotels, and the meetings will be held at the Statler Hotel, and Orchestra Hall. The Hospital Standardization Conference will extend from Monday morning to Thursday afternoon and will include a discussion of hospital and nursing problems and hospital demonstrations. Monday evening's program will include an address of welcome by the local Chairman, the address of the retiring President, the inaugural address of the new President, and the John B. Murphy oration. Clinics in general surgery will be held in the Detroit hospitals each morning from Tuesday to Friday, and the Eye, Ear, Nose and Throat work the same afternoons. Clinics will also be held at University Hospital, Ann Arbor, Tuesday to Thursday. On Tuesday and Wednesday mornings and afternoons, and on Thursday morning, clinical demonstrations will be held at the Statler Hotel (mornings) and Orchestra Hall (afternoons). On Thursday afternoon the annual meeting of the Governors and Fellows will be followed by a cancer symposium. On Friday afternoon there will be a symposium on traumatic surgery, to be participated in by leaders in industry, labor, indemnity organizations, and the medical profession. On Tuesday evening the program will take the form of a celebration of the Lister Centennial. On Thursday evening there will be a large Community Health Meeting in the Masonic Temple, and on Friday evening the Annual Convocation of the College. Other outstanding features will be the exhibits. In addition to the commercial exhibits there will be a replica of the Lister exhibit at the Wellcome Museum of Natural History, London, including Lister's operating rooms and hospital wards. The Departments of Hospital Activities, of Literary Research, and of Clinical Research of the College will also present exhibits. Among the foreign guests will be Sir John Bland Sutton, England; J. M. Munro Kerr, Scotland; Gordon Craig, Australia; Gustav E. Essen-Moller, Sweden; S. A. Gammeltoft, Denmark. The retiring President is W. W. Chipman, Montreal, and the President to be inaugurated, George David Stewart, New York. The Lister oration will be delivered by W. W. Keen, Philadelphia. The Chairman of the Detroit Committee on Arrangements is Alexander W. Blain.